ARCHIVES OF OPHTHALMOLOGY.

REPORT OF A CASE OF VESSEL-FORMATION IN THE VITREOUS

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(With one illustration on Text-Plate IV.)

THE following case of vessel-formation in the vitreous has some interesting features.

Mr. W., aged 26, noticed before his right eye, on the night of December 31, 1910, "a streak" to which he gave little thought. The next morning "the smooth, whitewashed plaster" of his bedroom "appeared to be rough and specked." When first seen, Feb. 11, 1911, the condition of the right eye was as follows: V. = $\frac{18}{10}$, imperfectly. The vitreous contained, besides the peculiar vascular fibrinous masses to be described, a multitude of fine, dust-like opacities, which, however, did not conceal the fundus nor interfere materially with the transparency of the vitreous. Pupil, iris, anterior chamber, lens, sclera, conjunctiva, and nerve head were all normal. The retina and retinal vessels, save for the exceptions to be noted, were apparently also normal. The iris dilated well under atropia. There were in the vitreous two highly vascular, glistening, transparent, fibrous masses—one of these in the upper half of the vitreous, the other in the lower-outer portion. The summits of these masses were to be measured by an 8 D. lens. The one in the upper half of the vitreous was very large, together from side to side occupying two thirds of the extent of the vitreous in that region. This mass consisted of a delicate transparent and semi-transparent, whitish and glistening fibrinous exudate, within which were great numbers of blood-vessels. These varied in size from vessels as large as the largest retinal arteries to vessels too small to be clearly made out under the examining lens. They anastomosed extensively. The mass in the upper part of the vitreous was made up by the mixing

together of three large fibrinous masses whose vessels anastomosed. This collection, or these collections of fibrinous exudates, proceeded from five long stalks, or pedicles, four of which sprung from the two main branches of the upper retinal vein—one from the temporal and the other three from the nasal branch. The fifth sprung from another temporal retinal vein. These stalks passed upward through perfectly transparent vitreous, the retina being normally visible beyond them in all directions. At first glance these pedicles appeared to be venous trunks directly continuous with the retinal veins; closer examination, however, showed them to be devoid of a light streak, while the smallest of the five could be seen to be only a cord of non-transparent, fibrinous material. At two places where these pedicles leave the upper nasal vein (*vid.* illustration) the vein could be seen to bend at an acute angle into the vitreous but to return at once to its bed in the retina. The largest of the five cords leaves the upper nasal vein at such an angle as to make it impossible to say whether this vein also is drawn upward into the vitreous. This, however, is probably the case. This stalk is continuous with the largest system of vessels seen in the fibrinous mass. Most of the vessels in these masses are merely minute threads in size, but under the examining lens are large enough to be seen clearly as vessels. The fibrinous mass in the lower-outer portion of the vitreous was much smaller than the one in the upper part. This was connected by a long fibrinous pedicle with the outer part of the lower temporal retinal vein. It was so situated that its every part was clearly visible under the examining mirror. Where the pedicle left the vein, the latter bent into the vitreous (*vid.* illustration) to return, after making an acute angle, to its retinal bed. In this mass the vessels were very numerous but of the smallest size visible to a + 8 D. lens. The vitreous about these masses was perfectly transparent, save for the minute opacities mentioned above as being general in their distribution. In the left eye, in which "the sight had been failing for nine months or more," the vitreous was so filled with exudate as to reduce the vision to counting fingers indistinctly at four feet, and to prevent any satisfactory examination of the post-lenticular regions. An orchitis told of the probable nature of intraocular changes. Mr. W. was put upon mercury and iodide of potash. By March 20th he was taking of the latter 285 grains three times a day. On this date the vitreous, O. D., was almost free of the dust-like opacities and the vision had risen to $\frac{1}{18}$. I could make out no change in the details of the fibrinous masses. In the fundus, other than mentioned above there was nothing of

interest save a minute pinhead-sized hemorrhage into the retina just beyond the origin of fibrinous stalk from the inferior retinal vein. The left vitreous had so far cleared, that there could be made out large masses of fibrinous exudate similar to those in O. D. These contained multitudes of vessels. There were, however, still so many large opacities in the vitreous as to prevent a detailed picture of these masses. The fundus was still invisible. The iodide of potash was continued. On April 17th the following notes were made. The masses in the vitreous appear to contain now only the faintest amount of exudate. The vascular elements have, however, much increased and outshoots from the parent masses can be seen to be extending in many directions. These outshoots consist of thin webs of vessels which are directly continuous with those in the masses. The retinal veins at the points of emergence of the stalks are pulled distinctly farther upward into the vitreous. In one place one of the stalks seems to rise into the fibrinous mass and then to descend again farther towards the periphery. These stalks look more like true retinal veins than do any of the others and highly suggest that this branch of a retinal vein has been pulled bodily upwards into the fibrinous mass. A small mass of highly refracting fibrin prevents the accurate following of the posterior course of this stalk. A small hemorrhage can now be seen in the retina external to the macula.

The above case suggests several things of interest. It seems fairly evident that the fibrinous masses came originally from the retinal veins and travelled along the routes marked out by their pedicles.

It is worthy of note that the retinal veins bend forward into the vitreous at the points of origin of the pedicles for these masses. It is as though the masses in contracting pulled upon the pedicles and these in turn pulled the veins forward into the vitreous. Then comes the question as to the origin of the blood-vessels in the masses. Are these vessels all veins or are they both veins and arteries? It is to be noted that all of the six pedicles are attached to retinal veins—not one to an artery. Although some of these vessels are very large I could not make out whether they were veins or arteries; they resemble in some respects both. What is the necessity for their existence and their great development in these fibrinous masses? If any of the vessels are arteries, whence their origin? If there are

no arteries, how is the circulation of this vast network of vessels kept up? The fibrinous masses lie free in the vitreous, being separated everywhere from the fundus by a considerable area of fibrinous-free vitreous. The stalks or pedicles connecting the retinal veins with the fibrinous masses are apparently cylindrical in form and are about the size of the veins to which they are attached. They are not transparent, as are the fibrinous masses. There is nothing to suggest any dilatation of the veins at the points of their origin. If they contain any blood-vessels these latter are so small that I was unable to make them out and yet, as mentioned above, the largest pedicle to the upper mass is continuous with the largest system of vascular branches in the mass. It is to be noted that nowhere in the fundus are there evidences of old or fresh hemorrhages if we except the two small spots above mentioned. The retinal vessels, arteries and veins, are, save at the points of origin of the pedicles from the latter, as far as can be seen under the ophthalmoscope, perfectly normal. The normal vision shows how little the general retinal substance partakes of the local disturbances. It seems remarkable that there are no evidences of hemorrhage about the origins of the pedicles from the veins. This seems to tell of the existence of a focal perivasculitis of a mild chronic nature. The fibrinous masses would then represent the result of nature's effort to repair the threatened breach in the walls of the veins. The antero-posterior currents in the vitreous would account for the carrying of the fibrinous exudate toward the front of the ball. It would seem to be necessary that there should be a vascular connection between the veins and the fibrinous masses, and that this connection should be along the pedicles. This, however, will not answer the question as to the necessity for an arterial element in the circulatory system of the fibrinous masses. I see no way to answer the question unless we suppose the disease to originate in the vasa vasorum system for the retinal veins. Granted that the smallest vessels are capillaries this does not do away with the necessity for an arterial element.

May 15, 1911. Changes in O. D. are noteworthy. Further enlargement of the fibrinous mass in the lower

outer part of the vitreous, considerable increase in the number and size of its blood-vessels, increase also in the size and number of the blood-vessels in the larger mass. These are forming new loops in several directions. The retinal vein from which springs the pedicle to this mass is pulled farther upwards into the vitreous at the point of attachment. The retina in its upper half shows extensive streaking and other evidences of irregular œdematous infiltration, with two or three small hemorrhages. Fundus O. S. still invisible, although there is marked increase in transparency in the anterior third of the vitreous, projecting into which can be seen great numbers of large blood-vessels in every way similar in appearance to those in the fibrinous masses visible in the vitreous O. D. May 16th. Patient given, intravenously, full dose of 606. June 15th, O.D., still further increase in the vascular loops growing from the smaller of the two masses in the vitreous. These finger-like projections in which one or more small anastomosing vessels can be plainly seen are confined to no one part of the periphery of the fibrinous mass. Increase in number of these projections from the larger fibrinous mass is less marked. The small hemorrhages in the fundus have disappeared, as has the œdematous appearance of the retina. Vision remains $\frac{1}{10}$. O. S.: Here great changes have taken place. The exudates into the vitreous have entirely disappeared save those in the immediate vicinity of the large fibrinous mass (to be later described) and centrally in the vitreous. The fundus is now everywhere visible and shows no changes in the retina or nerve. The vision is now with a $-\frac{1}{2}$, D 180° = $\frac{1}{20}$. The upper temporal retinal vein at a point almost above the macular region is pulled forward into the vitreous; from the angle thus formed proceeds outwards a grayish cord about the size of the diameter of the vein. This cord ends anteriorly in a mass of fibrin similar to the two described as present in the vitreous of the right eye. This mass presents the same large and small vessels and the same projecting vascular fingers. It differs from those in the right in that, free in the vitreous about the mass are numerous small grayish inflammatory masses of exudate. The amount of fibrin in the large mass is considerably greater than that present in the masses of the right eye. The rapid clearing of the vitreous may be attributable to the use of salvarsan, although Mr. W. has taken in addition 240 grains of iodide of potash three times a day. The opacities in the central part of the vitreous are not of the dust-like variety, but appear as small flakes of irregular size. Oct. 14th, O. S., vision reduced to counting fingers at six inches. Vitreous again filled with an exudate, and gives a blackish reflex. No details of

fundus or condition of the vitreous possible. The sight of this eye had remained good until the evening of Aug. 28th when it more or less suddenly "went out." At the same time "the vision of the right eye became worse, remained so for a few days, and then seemed gradually to return." Examination of the interior of the right eye shows no visible changes in the fundus. The masses in the vitreous are unchanged save that the vascular projections have increased in extent and a tongue from the upper larger one now reaches almost to the upward projections from the lower smaller mass. One of the larger protrusions from the lower mass gives off several finger-like projections around which the blood-vessels wind spirally, giving to these projections a much darker appearance than is possessed by the other projections from the masses. One thing seems clear—neither mercury and large doses of potash, nor the salvarsan have been able to stop the progress of the new formed vessels in the vitreous, and the eyes seem doomed.

Jan. 24, 1912. Vision O. D. excellent. Projections from fibrinous mass in lower half of vitreous have extended upward until they have reached the downward projections from the upper masses. The vascular limbs have increased in all directions, until now they exist in great numbers. Since last examination the tendency for many of the branches to end in knobs made by winding of the vessels around themselves, at their extremities, has much increased. The lower temporal vein at the point from which the stalk proceeds is now pulled forward at an acute angle into the vitreous to an amount to be measured by 3 D. At one place in the vitreous there proceeds from one of the vascular stalks a broad flat sheet of anastomosing vessels with numerous extending fingers. The other stalks shoot upwards branching and rebranching like miniature leafless trees whose twigs all anastomosed. In the left eye, whose vitreous is once more free of exudate save for the fibrinous masses about the vascular bodies, it can be seen that the retinal substance along the region of the upper temporal vein and artery is slightly detached by the presence of exudate beneath. In this eye the vascular body extends almost to the posterior surface of the lens. Several points of interest developed between October and January; of these the pulling of the retinal veins forward into the vitreous at the points of origin of the stalks and the increase in the vascular projections are most worthy of comment. As mentioned above, in the right eye the lower temporal vein as the result of this traction is pulled forward until it makes an angle of 30° with itself. In the left eye the retina along the course of the upper temporal veins is detached and at the point of emer-

gence of the stalk the vein is pulled forward almost at right angles to its course. This unusual condition must be the result of the contraction of the fibrinous material in the vascular bodies which are connected with the veins by the stalks and seems to show that the stalks themselves consist largely of new formed connective-tissue and are participating in the contraction. The vascular heads to the stalks are interesting. They have slowly but constantly increased in size ever since the patient has been under observation, and while they now reach almost to the posterior surface of the lens, they have not extended so as to lie directly behind the pupil. The patient's vision accordingly remains excellent in the right eye, and the usual examination of the nerve and macular regions would reveal nothing to make me suspect the presence of these large vascular areas.

Twice since the patient has been under observation the left vitreous body has been so full of exudate as to give a dark reflex and to preclude any picture of the fundus or the details of the contents of the vitreous. At present the vitreous is perfectly clear, save for the white highly refracting masses of fibrin and the vascular trees. There are no deposits in the fundus except that which lies subretinally along the region of the upper temporal vein. The patient has been almost constantly under mercury and potash—of the latter he has taken a large quantity. What acts as a stimulus to the continuous formation of the vascular offshoots is probably the presence in them of the S. pallida; otherwise it would seem that these minute vessels should atrophy or at least remain stationary. The eyesight is threatened from two directions—the traction in the retinal veins due to contraction of the fibrinous tissue within the vascular masses and the further spread of these masses. The best description of vessels of new formation in the vitreous which I have been able to find is in Loring's text-book of *Ophthalmology*, p. 40. Nowhere, however, have I found a description of a case whose details were the same as those in the one I have tried to describe. The illustration of the conditions present in the right eye on Jan. 25, 1912, is necessarily imperfect owing to the difficulties in reproducing the appearances at different levels of the vitreous and of giving the draughtsman any adequate idea of the ramifications and anastomosing of the minute and larger vessels as they appear under the magnifying lens. As one looks down upon the

vascular masses they give the appearance of a many-limbed, leafless tree whose small branches anastomose freely, while many of the terminal twigs ended in a knob formed by the twigs, winding around itself. Many of the vessels in the masses are much larger than is shown in the drawing.

THE SKIASCOPIC TEST. A NEW AND ORIGINAL EXPLANATION.

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(With seven figures in the text.)

THE principle upon which this explanation of the movement and locality of the shadows of skiascopy is based is that of regarding the emergent rays from the observed eye as all important. The great majority of authors lay too much stress upon the incident rays,—upon the course followed by individual rays from the source of illumination to the mirror, and thence to the patient's retina, laboring meanwhile over the incidental refractive error. This latter is of negligible importance to the incident rays and can be dismissed in a word. Its sole function is to convert the theoretic "point" of illumination upon the retina into an area, or region, by virtue of the diffusion circles created, and, as we shall see, this is a factor of neither inconvenience nor instability in the appended explanation.

USING A PLANE MIRROR.

Rays of light from the source of illumination (the dark-room lamp) are reflected as divergent rays, entering the observed eye as though coming from a point behind the mirror (see Fig. 1). If the mirror be tilted *up*, an area of retina is illuminated which lies *higher* in the vertical plane than the area of the preceding mirror-position. (See Fig. 1, A and A^1 respectively.)

USING A CONCAVE MIRROR.

Divergent rays from the source of illumination falling on a concave mirror are so reflected as to become convergent,

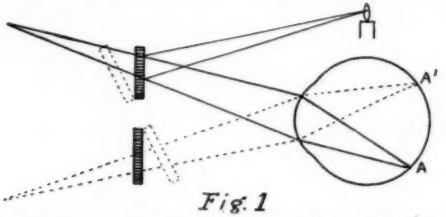

Fig. 1

and meet to form an aërial inverted image of the lamp; and this aërial image becomes the true source of retinal illumination (see Fig. 2). On tilting the mirror *down*, the true source

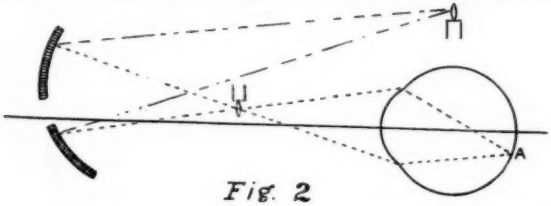

Fig. 2

of illumination comes to lie at a lower vertical plane than its forerunner, and the retina is illumined from this aërial lamp at a *higher* vertical-plane point (A') than was the case with the first mirror pose (A). (See Fig. 3.) Note that the two

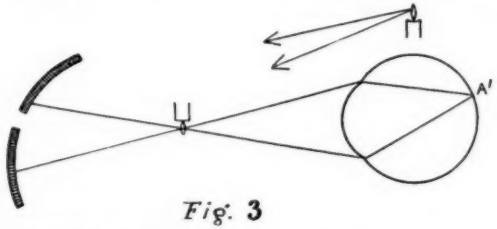

Fig. 3

mirrors, therefore, throw the reflected rays upon the retina in converse altitudes.

Of course, a similar case holds for retinal latitude, as the mirror is deflected to R or L.

Having determined the manner in which the retina is illuminated, it now becomes urgent to regard these areas as reflecting surfaces, the fountain sources of *emergent* rays. Reference will be made to a point as such source, but be it understood that this is merely for convenience and to reduce the complexity of diagrams; an area might be sketched in and would but serve to make the cones of rays larger and less defined.

HYPERMETROPIA, PLANE MIRROR.

Let the mirror be slightly tilted down so as to illumine a "point" just *below* the horizontal plane.

See Fig. 4. From point A of observed eye (the point of illumination with the mirror slightly downward tilted) emerges a series of luminous rays, a cone of which escapes through

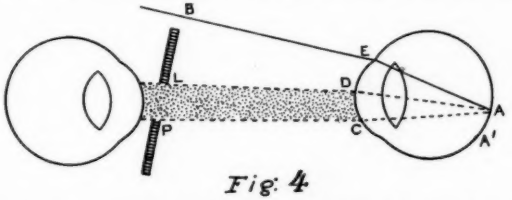

Fig. 4

the pupil and assumes a divergent course, in accordance with the fundamental property of the hypermetropic eye. Some of these rays enter the observer's pupil: (P–L) behind the mirror; others are lost to him (L–B). Tracing back the rays P–L, it will be observed that these come from the lower portion of the observed cornea (in reality, the pupil) (C–D), whereas L–B come from the upper portion (D–E). Now as no rays from the area D–E enter the observer's pupil, the region is blank to his visual perception; it is therefore dark —in short, it is the "shadow."

Further depress the mirror and a point more peripheral than A is illumined (A'), whereby the emergent cone of rays is directed higher than previously; B–L increases at the expense of P–L; in other words, D–E spoliates D–C; the shadow lengthens by creeping down the observed cornea and diminishing the luminous region. *The shadow has moved "with the mirror."*

Closer study will convince the reader that it is the observer's pupillary border which gives to the shadow its crescent shape.

EMMETROPIA.

Precisely the same principle and explanation suffice, with the exception that the cone of divergent rays of the hypermetropic eye must be modified to constitute a cylinder of parallel rays; and herein lies the explanation of the so-called hypermetropic shadow of emmetropia.

MYOPIA.

Let the mirror again be turned slightly down so as to illumine a "point" just below the horizontal plane.

A convergent cone of rays emerges, meeting at the punctum remotum, whence they proceed again as a cone, but of divergent rays, towards the observer's pupil.

See Fig. 5. In the present instance the packet of rays L–B passes out of range of the observer's pupil, and therefore the area from which it issues has no corresponding area

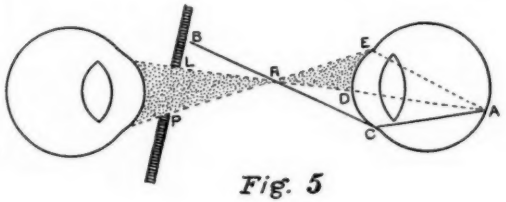

Fig. 5

of retina stimulation in the observer's eye. Tracing L–B back to the observed cornea, it is seen to issue from area D–C, which is, therefore, the "shadow," D—E again being luminous. Observe that the mirror was down-tilted and in result the upper part of the cornea again shaded.

Proceed to a more pronounced down-tilting, which serves to carry A towards the periphery and thereby to throw the divergent cone to a higher vertical level, so reducing the luminous allotment PL–DE simultaneous to the increase of the shadow area C–D. The shadow has therefore moved up when the mirror was depressed,—*it has moved "against the mirror."*

MYOPIA I D. OR LESS.

The converging cone will have its focus (R) somewhere beyond or at the observer's eye, he being stationed at one metre from patient's eye. The shadow will then move with the mirror, as can be demonstrated by figures sketched upon the above principles.

CONCAVE MIRROR.

As explained above (see Figs. 2 and 3), the downward (or lateral) tilting of the mirror functions to illumine a retinal area, or "point," in the converse meridian.

HYPERMETROPIA.

Downward tilting of the mirror passes A above the mid-horizontal plane of the eye, in consequence of which the divergent cone of rays issues with its axis of direction inclined downwards toward the floor.

Reference to Fig. 6: illustrates the luminosity of the

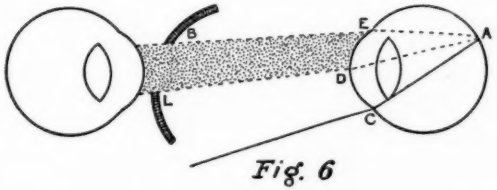

Fig. 6

segment D–E, and the blackness of segment C–D. The farther A approaches the periphery of the fundus, by so much more is the cone depressed; equally so is D–E reduced, and co-equally does C–D magnify: the shadow creeps upwards upon the observed cornea, as the mirror is depressed; it ***moves against the mirror.***

MYOPIA.

Reference to Fig. 7: By down-casting the mirror, A is illumined and the area D–C of observed cornea shows the red reflex, the area D–E being the shadow. As the mirror is lowered, so does the cone B R P depress; simultaneously the

luminous area B–L is reduced, from L; in other words, the area D–C of corneal luminosity shrinks from D towards C; which again may be translated to terms of E–D (the shadow)

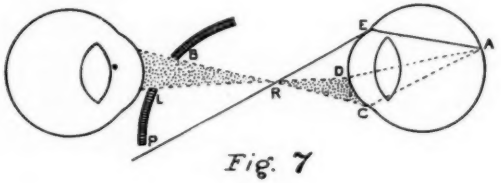

Fig. 7

encroaching more and more upon D–C: the shadow increases from above down; it ***moves with the mirror.***

Slips of paper cut to the shapes of the emergent cones of rays, moved round the fundus, will assist not a little in rendering the increase or decrease of the shadow, or the luminous pupil, more facile of comprehension.

THE EYE SYNDROME OF DEMENTIA PRÆCOX.[1]

OCULAR SIGNS AND SYMPTOMS OF DEMENTIA PRÆCOX AND THEIR SIGNIFICANCE, AS OBSERVED IN 115 CONSECUTIVE CASES.

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(*With three colored drawings on Text-Plate.*)

IN 1899, Siglas saw a case of anxious melancholia, followed by mental confusion, evolve parallel with gastro-intestinal autointoxication. Meyer reported five similar cases. The idea that dementia præcox is an autotoxic disease originated with Morro, who first connected hebephrenia with this cause in 1900. The evidence for its autotoxic nature may be grouped as follows: (1) There is a coincidence of certain ocular symptoms with gastro-intestinal autointoxication similar, in many aspects, to those seen in typhoid, lead colic, and simple intestinal putrefaction; (2) the urine in dementia præcox shows very defective elimination; (3) fully one-half of the subjects of dementia præcox die of tuberculosis; (4) the coexistence of certain toxic dermatoses, such as certain types of erythema, vasomotor paresis, with chronic gastro-intestinal

[1] This work was undertaken at the suggestion of Dr. Clark, and the greater part of the work was carried out in his service at the Manhattan State Hospital and at the Vanderbilt Clinic (Dr. Starr's service). Dr. Tyson made the eye examinations and is responsible for the detailed findings of the same. Both authors are jointly responsible for the interpretative significance of the study.

intoxication, is noteworthy; (5) some additional facts are at hand, as shown in the co-existence of psychic excesses, neurasthenia, etc., with states of autointoxication; (6) the study of the blood in dementia præcox shows evidence of a toxic state.

More specific evidence is at hand in Kuhnt and in Blin's work, especially the latter. In 1905 Blin published a monographic consideration of the autotoxic nature of dementia præcox. He attempted to show that the retinal changes are analogous to those seen in various acute and chronic infections. It is of passing historic interest to say that Dide and Assicot (1901) had already noted the alteration of anæmia and congestion in the disks of dementia præcox. None of these various observers have made any thorough or systematic attempt to analyze the significance of the eye changes in dementia præcox.

The subject of the ophthalmoscopic changes in dementia præcox receives little attention in the most recent reference works. Our bibliographic research has unearthed but two studies in which the subject is considered. These are the general papers by Kuhnt and Wochenin on alterations in the retina in psychoses, and the work by Blin on the ocular changes in dementia præcox. Kuhnt and Wochenin[1] examined 511 cases of mental diseases with the ophthalmoscope. Of these there were 5 cases of hebephrenia. The other forms of dementia præcox are not mentioned. Pathologic changes in but one case are mentioned, presumably the others were not noteworthy. This one patient had pallor or anæmia of the temporal half of the papilla.

The work of Blin is much more to the point. He examined the retina in 87 cases of dementia præcox. A second examination was frequently undertaken. Despite his extensive material, Blin appears to have made no careful analysis of the significance of the findings. Of the 87 cases some abnormality of the papillæ was found in 59. The material was divided into three groups. Nine cases (10.2 per cent.) showed constant hyperæmia of the disks, and in 23 (36.8 per cent.) the congestion was transitory. Blin does not mention the condition of the blood-vessels as to overfilling, tortuosity, etc., nor does he appear to have examined closely into the coincident

[1] "Ueber Veränderungen der Netzhaut bei Geisteskrankheiten," *Ztschr. f. Augenh.*, 1903, xiii., 89.

eye symptoms necessary to establish an eye syndrome for dementia præcox. An anæmia of the papillæ was constant in 7 cases (8 per cent.), and inconstant in 15 (25.3 per cent.). He uses the term intermittent in connection with the latter. There were 5 cases in which congestion alternated with anæmia (5.74 per cent.).

Various points often mentioned in individual cases are not summed up, such as haziness of the border, or the absence of demarcation between papillæ and retina, predominance of lesion in one eye, and the like. Doubtless all these facts seemed somewhat contradictory and did not lend themselves easily to a summary.

Three years ago we undertook an independent research on the significance of the ocular signs and symptoms in dementia præcox. We have analyzed 115 consecutive cases. The work was undertaken with the view that a careful analysis of the eye symptoms in dementia præcox might throw some definite light on the uncertain and perplexing pathogenesis of the disorder. In this respect, we believe we have not been disappointed, inasmuch as we have found definite changes and symptoms in all cases which are fully distinctive of this psychosis.

The fundus changes as seen clinically may be divided into three groups, which are usually in the order of their occurrence, as follows:

1. Congestion of disks; hyperæmia and œdema; dilated, dark colored veins; slightly contracted arteries and blurring of the edges of the disks, all varying in degree. These changes constitute a low grade of perineuritis of the optic nerve.

2. Congestion of the nasal side, with temporal pallor of disks, dilated veins, contracted arteries.

3. Pallor of disks, dilated veins, contracted arteries. These changes constitute anæmia and partial atrophy of the optic nerve.

One hundred and nine cases were examined with the ophthalmoscope; 55 were males and 54 females. The ages of the males were from 12 to 47 years, and those of the females were from 13 to 39 years.

All the different forms of dementia præcox were under study. While the results by form-types have not been fully analyzed.

we are prepared to say that the more marked changes in the eye syndrome were found in the more rapidly deteriorating types of dementia præcox.

The cases embrace those who have used alcohol and tobacco moderately or to excess, as well as abstainers. It is possible that alcohol and tobacco have contributed toward the clinical picture in some cases. In differentiating the pathologic condition of the optic nerve in tobacco- and alcohol-users from those in cases of dementia præcox, one observes central scotoma in the former. The one case showing a central scotoma for red in dementia præcox gave a history of alcohol and tobacco excesses. The disk changes in dementia præcox have some resemblance to those seen in the toxic amblyopia of tobacco and alcohol. But the fundus changes above detailed are seen in all cases under study. There is strong evidence that some other potent toxin is responsible for the disk changes in dementia præcox. We do not hesitate to say that we believe the toxin is primarily a vascular poison. Its most probable source is in the autointoxication (intestinal putrefaction is almost invariably evident from clinical symptoms) from the intestines or from the liver. It is possible that a faulty metabolism from a perverted action of some of the ductless glands (thyroid especially) may be the pathogenic agent. The primary departure from the normal in the disk is in the veins. They become dilated, tortuous, and darker than normal. Œdema of the disk appears shortly afterward. These changes are analogous to those seen in the passive congestion of the face and hands in dementia præcox cases. All these œdemas produce such lasting disturbance in the nutrition of the optic nerve that slow degeneration of the nerve-fibres finally results. Thus, of the 109 cases examined by the ophthalmoscope, a low grade perineuritis was found 62 times in the right eye and 67 times in the left. Temporal pallor, with nasal side congested, was found in the right eye 10 times, in the left eye 11 times. Pallor of disks was found in the right eye in 37 cases and in the left eye in 31 cases.

Inasmuch as the disk changes in the first stages resemble somewhat those seen in ordinary intestinal toxæmia, we have repeatedly examined the disks in a number of cases while the patients were under active hygienic treatment of free catharsis,

intestinal antiseptics, baths, and dietetic regulations. A marked degree of betterment was noticed of the congestive margins of the disks, but the central œdema and transitional pallor have continued. Indeed, while patients were under this treatment a general physical improvement was noticed, but the mental state seemed little improved.

Coincident with the study of the changes in the papillæ, the pupils were examined in 85 cases. The changes uniformly found here were not less significant. The examinations were made in moderately light rooms with the eyes fixing a distant object. The size of the pupils varied from $3\frac{1}{2}mm$ to $7mm$, with an average of $4\frac{77}{85}mm$, while the average of the control pupils (physician and attendants) was $3\frac{48}{85}$. An average enlargement of $1\frac{29}{85}mm$ for dementia præcox over the normal was evident.

The light reaction was active in 71 cases and sluggish in 14. Consensual reaction was active in 68 cases and sluggish in 17.

Accommodation and convergence were active in 71 cases and sluggish in 13. Hippus was present in one case.

The sensory pupillary reflex was slightly positive in 6 cases and negative in 79 instances. The psychic reflex was slightly positive in 4 cases and negative in 85 cases. Piltz-Westphal reflex was positive in 2[1] and negative in 85 cases.

Great care was exercised in measuring the pupils on account of the tendency of the eyes to change their visual lines. One is apt to complicate the accommodation and convergence reflex with the other tests. The negative reactions of the pupil appear to be due: (1) to loss or partial loss of function through defective nerve innervation; (2) defect in attention; (3) diminished apperception.

Corneal sensibility was diminished in 69 cases and normal in 17.

The visual color fields were examined in 81 cases. All were found concentrically contracted. The largest field was 30 degrees. The smallest 0 degrees. The fields were practically abolished. The average of the 81 cases was 10.6 degrees, which was a marked contraction from the normal. This may be explained partially by the inattention of those patients, diminished capacity for externalization, and finally, and not

[1] At first positive, later examination was negative.

least, by the œdema and congestion of the optic nerve (perineuritis) in the first stage of degeneration of the nerve and by the ultimate shrinkage of the new connective-tissue in the partial atrophy of the nerve.

The changes in the disks, pupils, visual fields, and corneal sensibility which, when taken together, constitute the new syndrome, are all in accord with each other. In our examination of all other types of insanity, imbecility, or idiocy we have found no other condition similar to what we have outlined here for dementia præcox.

The clinical significance of these findings is of importance:

1. They indicate that dementia præcox is attended by such an early and constant syndrome of alteration of disk, visual field, pupil, and corneal sensibility as to materially aid in diagnosticating this psychosis. Consideration of the syndrome will particularly aid in the differential diagnosis of dementia præcox from the manic-depressive group, acquired neurasthenia, hysteria, and the various forms of imbecility and constitutional inferiority.

2. The syndrome is a distinct contribution to the theory that dementia præcox is an autotoxic disease, and that the poison is primarily vascular, which finally induces neuronic degeneration. It points to a toxin of some sort, which is either a metabolic defect in the tissues (ductless gland defect) or, what seems more probable, that the poison is generated in the liver or in the gastro-intestinal tract itself.

3. The syndrome is of prognostic value, as the severer grades of eye changes are found in the more rapidly deteriorating cases.

4. Finally, the optic-nerve lesion is quite in accord with our best knowledge of the pathologic anatomy of dementia præcox, in other tracts of the brain (than the optic nerve which itself may be counted an analogue). The early vascular changes in the brain ought to receive more serious investigation.

We desire to thank Dr. Mabon and his staff, Dr. Smith of Central Islip and his staff, and the staff of assistants in the neurologic department of the Vanderbilt Clinic (Dr. Starr's service), and Dr. C. E. Atwood in particular, for the courtesies extended to us in placing their patients at our command.

Since the publication of our original paper upon the eye syndrome of dementia præcox no similar carefully recorded observations upon the subject have been made. It is true, however, that the fundamental postulate of the cause of the syndrome, namely, the toxic character of the mental disorder, has been under continuous study. The rival camps of a psychic or somatic origin for the disease have been in a constant state of warfare. The attitude of the Kraepelinian school of somatists and the Meyer–Hoch school of psychogenists are too well known for us to detail the special viewpoints of each here. It is hoped a mutually supportive middle ground may succeed the present-day partisanship.

The main contention of our paper is that a fairly constant eye syndrome exists in dementia præcox and secondly the best hypothesis for explaining the presence of the same is upon some endogenous or exogenous toxic substance. We believe the first position has been established. From the partial and incomplete nature of our study the second contention can only be urged as a contributing study to that end.

It is unnecessary to point out the remarkably low resistance of nerve-tissue to toxins in dementia præcox. As is well known in ophthalmic practice, degenerative changes in the nerve head from toxins are at first transitory, requiring time for producing distinct pathological changes as shown exquisitely in tobacco amblyopia. Even then such fundus alterations are apparently slight and not recognizable microscopically but should be by more delicate methods of analysis. The extra vulnerability of nervous tissue in dementia præcox may be fairly well shown in a kindred state of inherited vice of constitution, that of imbecility. In a study of the eye in mental defectives by Clark and Cohen,[1] one of the objects of the study was to note whether a parallelism in constitutional defect, namely a strong tendency to neural degeneration, existed in both disorders. It is interesting to note in this connection that the authors found fully three-fourths of all cases of idiocy of their study showed varying degrees of retrobulbar neuritis of a degenerative character. However one may search for exciting causes in the degeneration of the optic nerve in idiocy

[1] "A Study of the Eye in Mental Defectives," *Journal of American Medical Association*, April 16, 1910, vol. liv., pp. 1287, 1288.

or dementia præcox, one needs to bear in mind that parallelism probably extends no farther than an inherent tendency in both, for the development and character of the changes in each are totally unlike. Then, too, defectives never have the pupillary or other signs of the eye syndrome of dementia præcox.

In examining the fundus of the eye, allowance must, of course, be made for each individual variation within relatively wide physiological limits. The difference in appearance of the fundi in anæmic individuals and well-nourished ones is not inconsiderable. The same relative disproportion in sizes of arteries and veins exists in most all cases while, the actual calibre of vessels varies greatly.

As to the different stages of the fundus changes since studying our cases over a longer period of observation, it is a question with us whether our first and second stages should not be transposed. As is well known and first pointed out by Horsley, in choked disk from brain tumor the earliest changes are found in haziness of the superior nasal quadrant of disk. But as in the examination of the early cases examined at the clinics (pre-hospital or pre-asylum stage) we noted a majority of the congestive stage, *i.e.*, entire disk congested, etc., as originally described in stage No. 1.

Size of pupils: Those which were less than the normal average were taken in bright light and were included so as to account for and make a total average for all our cases examined, otherwise if the average size of pupils were considered only of those that had been measured in a moderately lighted room, as the majority had been, then the average size would have been a trifle larger than reported.

Great care needs to be exercised in testing sensibility of cornea; one should not touch eyelashes, nor lids, nor have image of approaching objects or test fall on visual field. One should carefully approach the cornea from the periphery. Additional cases making 200 in all have been examined and about the same percentage of changes has been found as was observed in the 115 first reported.

In examinations of fundi of dementia præcox cases by Dr. Holden, subsequent to our examinations, changes were noted by him in about fifty per cent. of cases, but he did not study the

syndrome in its entirety. He confined himself to examination of fundi alone.

The recent, careful, and rather exhaustive work of Southard upon dementia præcox is worthy of more than passing notice. Southard made a careful examination of 63 brains obtained from dementia præcox patients who died at the Danvers (Mass.) State Hospital. In his investigations he laid particular stress on the "topographic idea" which had occurred to him, when, some years ago, he made an analysis of the first 1250 necropsies of the Danvers State Hospital and collected the lesions of different parts of the brain in card catalogue form. In a surprisingly large number of cases he found local areas of sclerosis, atrophy, or aplasia. His conclusions in regard to dementia præcox are as follows:

"1. Existent evidence for the organic nature of dementia præcox is not wholly convincing, since (*a*) the cytologic changes described are found also in cases of toxic deliria and in cases complicated by severe visceral disease, and (*b*) the stratigraphic changes described are found also in senile cases without characteristic symptoms of dementia præcox.

"2. Resort must, therefore, be had to the topographic idea, for the adequate exploitation of which total brain sections, with cytologic exploration of *all* areas, are ideally necessary.

"3. Random blocks of brain tissue with demonstration of satellitosis, infrastellate gliosis, or disintegration products of cell disorder will throw little light on the mechanism of dementia præcox.

"4. The data of the functionalists (dissociation, sejunction, intrapsychic ataxia, and the like) are of the utmost importance as indicating the essential focality of the pathogenic process and the focal variations in its severity.

"5. The curability of certain cases, the remissive character of some cases, the speedy disappearance of particular symptoms, the persistent complexity of reaction in some instances, the absence of characteristic severe projection-system symptoms, all indicate that the process is histo-pathologically mild, and that the focal changes found will be but slightly destructive or even irritative (in the sense of slight injuries readily repaired or compensated for).

"6. Grossly destructive lesions of a transcortical character

in Wernicke's sense might conceivably effect, *e.g.*, a permanent catatonic complex and doubtless will be found to do so occasionally; but the protean and progressive character of dementia præcox will exclude such transcortical injuries from playing a large part in the pathogenesis.

"7. The focal lesions to be sought for will doubtless escape macroscopic notice in many instances, since the volume of apparatus engaged in effecting very prominent symptoms is often slight and is spread very thin in numerous areas.

"8. Studies of the "soft brain" and of gliosis in epilepsy have proved, however, that even comparatively slight degrees of cortical gliosis can often be palpated at autopsy.

"9. Palpable glioses of a focal or variable character combined in numerous instances with visible atrophy and microgyria, have been found in over half the series under examination, in cases regarded as clinically above reproach and *not* subject to coarse wasting processes, focal encephalomalacia, cortical arteriosclerosis, or diffuse chronic pial changes.

"10. The frequent co-existence of several foci of sclerosis or atrophy in the same brain and the microscopic observation of milder degrees of nerve-cell disorder and gliosis in regions without gross lesions tend to the conception that the agent is more general and diffuse in its action than would seem at first sight, so that future research may well demonstrate that certain instances of coarse brain wasting and even of diffuse chronic leptomeningitis belong to the group (microscopic corroboration necessary for assigning values to focal variations).

"11. The microscopic examination of the residue of cases, in which gross lesions or anomalies were not described, shows the same tendency to gliosis and satellitosis in numerous instances and the same tendency to focal variations from gyrus to gyrus exhibited by the gross lesion group. These findings suggest that the minor gross lesions and anomalies of several cases actually escaped notice (the protocols, though drawn up with a certain system, are by various hands) at autopsy, so that the probable actual proportion of gross lesions is 68 per cent. If microscopic evidence is resorted to, the organic proportion in our series rises to 86 per cent.

"12. Several groups of cases were classified from the distribution of microscopic lesions, although the focal purity of these cases can often be brought in question from the results of microscopic examination (infrastellate gliosis and satellitosis also in macroscopically normal areas).

"I. Pre-Rolandic group, including a superior frontal-prefrontal sub-group of paranoidal trend.

"II. Post-Rolandic group, including (*a*) postcentra-superior-parietal (sensory perceptual) sub-group in which catatonic features are the common factors; (*b*) occipital sub-group.

"III. Infra-Sylvian group (too small for clinical correlations).

"IV. Cerebellar group (catatonic features).

"13. If these data find general confirmation, they will doubtless go far to unify discussion, since mild, variable, and progressive intracortical lesions, proceeding at different rates in different parts of the apparatus, and having the peculiar distributions indicated above would explain adequately some of the contentions of the dissociationists, while remaining not wholly inconsistent with Kraepelinian ideas.

"14. The frontal-paranoid correlation is in line with modern physiologic ideas, but it must be granted that the occipital and temporal regions, as elaborating important long-distance impulses, may well play a part also in paranoid states.

"15. The cerebellar-catatonic correlation is doubtless in line with some contentions of the Wernicke school, and obvious comments might be made in connection with the proprioceptive functions of the cerebellum (Sherrington).

"16. The postcentral-superior-parietal relations to catatonic symptoms are perhaps theoretically the most novel suggestion from the work, but here again the results are not consistent with modern physiology.

"17. The topographic study of dementia præcox brains, both gross and microscopic, is commended as likely to shed new light on the pathogenesis of certain symptoms, notably paranoidal and catatonic symptoms."

These conclusions of Southard's are given at length not only as they are undoubtedly the best summary of the "topographic study" of the præcox brain at present hand, but because the lesions of atrophy and sclerosis are surprisingly analogous for

that we note in the progressive degenerations in the nerve head itself. Our work might therefore be properly called a clinical contribution to Southard's "topographical histo-pathological study."[1]

[1] Our special thanks are due to Dr. Braun for his faithful drawings, in color, of the eye grounds in our cases. Drawings are a part of the original contribution.

SOME OCULAR MANIFESTATIONS OF SYPHILIS AND PARASYPHILIS OF THE NERVOUS SYSTEM.

By GEORGE H. KNAPP, M.D., Cincinnati.

EVEN in their incipiency syphilitic and parasyphilitic affections of the nervous system are manifested with such frequency by ocular symptoms as to entitle the latter to the most careful consideration. Certain of these symptoms, such as the unequal, irregular, sluggishly reacting pupils in early paresis or the Argyll-Robertson phenomenon in tabes, are quite constant, indeed almost pathognomonic. On the other hand, the ocular expressions of what is more properly termed nervous syphilis are most inconstant and diverse, yet it is by reason of this diverse, kaleidoscopic, and almost bizarre quality that the symptoms have a certain individuality suggestive of the luetic nature of the process in question.

Since the nature of the pathologic process is such in syphilitic disease of the central nervous system that good can be expected to result from treatment only when this is instituted in the beginning of the disease, the early appearance of ocular symptoms gives to them a very great importance. It is the purpose of the writer to emphasize the fact that certain symptoms occurring on the part of the eyes, far from being of local interest only, are of the utmost importance in the diagnosis of a class of cases of frequent clinical incidence early in the disease and before irreparable and permanent damage has been done.

Even though certain ocular symptoms be not of themselves conclusive evidence of syphilitic or parasyphilitic processes, they are encountered with such frequency in such conditions as to be highly suggestive, and in conjunction perhaps with other physical signs and the Wasserman test or a cytologic

examination of the cerebro-spinal fluid may lead to the proper diagnosis of an otherwise obscure case and the institution of treatment at a time when there is a reasonable hope that the pathologic process will be cured or arrested. If one may accept the opinion of so high an authority as Dana (*Journal Am. Med. Ass'n*, vol. 54, 1910), even the treatment of paresis is robbed of its traditional hopelessness if instituted early enough. In this luminous article Dana cites fourteen cases manifesting the clinical symptoms of paresis which improved under treatment and remained well for years. In substance Dana's views are that in certain instances paresis is of insidious onset and slow development, so that in some cases changes in the nervous system, so subtle as to escape ordinary attention, have been going on for years before the disease becomes sufficiently obvious for the patient to seek medical aid; that there is an underlying unity between luetic neurasthenia or so called pseudoparesis and paresis in the usual acceptation of that term, and finally that fatal cases of paresis result so because of belated treatment or because the luetic toxin is uncontrollable by usual methods of treatment and produces degenerative changes in brain cells perhaps themselves peculiar in being of an abiotrophic quality. The lesson to be deduced from a careful perusal of the histories of these cases of Dana's is the importance of early diagnosis and that treatment under such conditions offers a reasonable hope of retarding the progress of the disease or even arresting it altogether.

Pathologically intracranial syphilis manifests itself: (*a*) in the form of circumscribed gummata occurring for the most part on the convexity of the hemispheres but frequently also in the interior of the brain; (*b*) as a diffuse gummatous meningoencephalitis of rapid formation and affecting chiefly the base of the brain and region of the chiasm; (*c*) by certain characteristic alterations in the cerebral vessels notably endarteritis obliterans. Of the pathology of the parasyphilitic affections, tabes and paresis, it need only be said that they are essentially degenerative processes and the more advanced symptoms, such as ataxia, anæsthesia, and dementia, have their pathologic basis in neural destruction (Mott).

This diversity in the location and character of the pathologic lesion in nervous syphilis accounts for the wide range and

diversity of the symptoms. A gumma anywhere within the cranial cavity may be accompanied by optic neuritis (W. Aldren Turner). This neuritis is usually bilateral, of early appearance, rapid onset, and intense degree. An example of double optic neuritis probably resulting from increased intracranial tension in a case of cerebral gumma is furnished by the following case:

M. B., female, æt. 24, single, referred by Dr. W. R. Griess, consulted me, October 4, 1911, and gave the following history: She had been in good health until about two months ago when she began to suffer from severe headaches, vomiting, and giddiness. The headache was excruciating, vertigo was of such degree that she staggered in walking, and vomiting came on usually after taking food but without nausea. She applied for admission to a local hospital where she was told that her symptoms were due to "nervous prostration," but as her symptoms did not improve she consulted Dr. Griess. She had then in addition to the above symptoms a motor aphasia, the right arm and leg were paretic, and she complained of dimness of vision. The symptoms of an intracranial tumor were recognized by Dr. Griess and a luetic origin suspected. The headache and vertigo cleared up in a short time upon antisyphilitic treatment, but Dr. Griess thought it advisable to give her an injection of "606" and referred her to me for an examination of the fundus. Vision in both eyes was reduced to $\frac{20}{100}$, form field concentrically contracted, she was unable to distinguish red but called it yellow or tan color; green and blue were recognized. Pupils moderately dilated, light response somewhat sluggish. The disk looked somewhat pale, outlines hazy, and there was a slight exudate in the circumpapillary region. Veins engorged, arteries small. I advised against the administration of salvarsan in the presence of an optic neuritis and K. I. was continued in large doses. Six weeks later the patient was apparently free from all symptoms, the partial dyschromatopsia had disappeared, and vision was $\frac{20}{30}$ in both eyes not improved by any lens

While the above case exemplifies the occurrence of optic neuritis or more properly papillœdema from increased tension excited by the presence of a gumma, inflammation of the optic nerve (the neuritis descendens of Von Graefe) may also result by the extension of a basilar luetic meningitis as in the following case.

Mrs. R., æt. 65, music teacher, married, and the mother of two children; three miscarriages at third or fourth month. Husband has been a paralytic for a number of years. On October 1, 1911, stated that she had suffered from headaches for the past six months but latterly these had become much more severe; also complained of vertigo. Vision had become so poor during the last month that she was unable to follow her occupation. Vision R. and L. $\frac{20}{200}$, no dyschromatopsia, form field concentrically contracted in both eyes. Pupils equal, regular, moderately dilated, respond readily to light. Papilla of pinkish gray color, hazy outline, arteries small, veins somewhat engorged but almost of normal size.

Syphilitic basis of ocular symptoms inferred from the intense headache, history of repeated miscarriages, paralysis on part of the husband, and the absence of other causes of neuritis. Improvement almost immediate under treatment by K. I.; headache and vertigo disappeared, and on December 15, 1911, vision R. and L. was $\frac{20}{30}$ partly with + 1.25 D. sph.

Since one of the commonest positions for the occurrence of a basal gumma is the interpeduncular space and region of the chiasm, hemianopsia (chiefly bitemporal) is of frequent occurrence. Varying degrees of ophthalmoplegia are produced by basal meningitis involving the third nerve alone most frequently, less often the fourth and sixth. For anatomical reasons the fifth nerve is apt to be affected along with the fourth or sixth so that corneal anæsthesia or a neuroparalytic keratitis may form part of the clinical picture.

The more common ocular symptoms of early tabes are the Argyll-Robertson pupil in its typical form, or the pupil may still contract, though sluggishly, when stimulated by light; the pupils in some cases are unequal, irregular, and have a tendency to myosis; ocular palsies if they occur are apt to be of rapid onset and short duration; the disk may look normal and central vision be good, while the form field is found to have undergone irregular, concentric contraction, and the patient has perhaps a partial dyschromatopsia. Exceptionally optic atrophy precedes the onset of other symptoms for years.

In early paresis Dercum emphasizes the significance of a slight degree of amblyopia which may or may not coexist with some impairment of the color sense. But chief of the ocular disorders of paresis is a gradual and progressive internal

ophthalmoplegia (W. Julius Mickle). In detail the progress of this ophthalmoplegia may be divided into four stages according to M. Gilbert Ballet (quoted by Mickle, *Brain*, 1894). These are, if we consider one eye: (1) paresis of movements of reaction to light, (2) abolition of these movements, (3) abolition of reaction to light and paresis of accommodation, (4) complete paralysis both to light and accommodation. Nystagmus is occasionally observed. *These ocular symptoms sometimes considerably precede other manifestations* (Mickle).

Happily we are no longer entirely dependent upon inferences from clinical symptoms or upon the statements of the patient in arriving at a diagnosis in cases suspected of an antecedent luetic infection but have at our disposal two valuable aids in the Wasserman test and in the cytologic examination of the cerebro-spinal fluid. The Wasserman test is practically constantly positive in tabes and paresis and in many cases of active syphilis of the nervous system. Lymphocytosis in the cerebro-spinal fluid is practically always present in active syphilitic processes of the central nervous system, in tabes, and in paresis. Hence a patient with a positive Wasserman and lymphocytosis in the cerebro-spinal fluid has an organic involvement of the central nervous system which we may safely infer is either an active luetic lesion or a parasyphilitic process.

Having established the fact that the patient's ocular symptoms are of luetic origin, due cognizance should be taken of the fact that these symptoms have more than a local significance and that they may herald or presage changes in the central nervous system of the most serious and widespread kind. While some of the symptoms of nervous syphilis with which ophthalmologists are concerned are of favorable prognosis and yield readily to treatment, we should be mindful of the innate remissive tendency of nervous syphilis and not be content with a therapeutic result which simply leaves the patient free from the symptoms because of which he came seeking relief, but he should be kept under observation for an extended period of time and thoroughgoing treatment continued until the Wasserman test has been found repeatedly negative. Regarded in this manner a group of symptoms frequently observed by ophthalmologists is seen in its proper relation and we have discharged our full duty to the patient.

TUBERCULOSIS OF CONJUNCTIVA AND SCLERA, FOLLOWING REMOVAL OF A PIGMENTED PAPILLOMA OF THE CONJUNCTIVA.[1]

By LUTHER C. PETER, A.M., M.D., Philadelphia.

Lillian S., æt. 13 years, of Cuban extraction, consulted me in reference to a congenital brownish pigmented area in the conjunctiva of O. D., *2mm* from outer limbus. It was equilaterally triangular in shape, with base in, *2.5mm* long. The pigmented area moved freely with the conjunctiva and apparently was elevated little if any above the conjunctival surface. The family observed that for some months previous there was slight increase in the size of the patch, and on request of patient's sister, who is a physician, it was decided to remove the growth.

April 26, 1911, under cocaine anæsthesia, the pigmented area was removed without accident. Two silk stitches were introduced to close the conjunctival wound and left in situ 48 hours. On May 2d, the wound was healed, but there was considerable residual redness and slight thickening of the conjunctiva. On May 13th, the area of redness had increased to about *12mm* in diameter and elevation was more pronounced. The color was now salmon and one large vein led well back over the globe to the inferior cul-de-sac. There was no pain. Visual fields and eye-grounds were entirely normal.

The report, from the pathologic laboratory of Dr. E. J. Asnis, of the removed growth was "pigmented papilloma."

May 22d (*i. e.*, about one month after operation), the patch showed an increase in diameter and elevation and contained two foci of ulceration. To the palpating finger, the thickened area was decidedly firm and somewhat gritty.

After consultation with Dr. Wendell Reber, it was decided to remove a section of the diseased area to determine the nature of the process. Two sections were removed and the

[1] Read before the Polyclinic Ophthalmologic Society of Philadelphia.

examination of these sections by Doctors Rosenberger and Roddy showed a tuberculous process. Their report is as follows:

"The sections vary in size, those at either end of the series being smaller than those in the middle. The six sections from the centre of the series have practically the same size and appearance. The sections show a fibrous-tissue margin, one side of which is evenly infiltrated with small round cells. The rest of the field is composed of lymphoid, giant, and round cells, and hyaline and granular matter, lymphoid cells predominating. Six distinct tubercles are present. There is one near each pole of the section, one near the centre, and three close together extending across the width of the section midway between the central tubercle and one of the polar tubercles. The three which form a group are nearly equal in size and are somewhat larger than the other tubercles; they also show a relatively larger inner or degenerated zone. In the centre of each there is a pale, poorly stained zone in which a few granules and fragments of cells are seen. Surrounding this zone is a circle of lymphoid and epithelioid cells which are so dense and so much closer together than in the rest of the tissue that they form a typical histological tubercle.

"R. C. ROSENBERGER
J. A. RODDY."

A careful physical examination by Dr. J. A. Roddy, the attending physician, and a careful search for tubercle bacilli in the sputum, urine, and fæces failed to reveal any foci of disease other than the eye. There is a history of tuberculosis in both maternal grandparents and one maternal aunt, but otherwise the family history is negative. The previous history of the patient is good, mumps and measles being the only diseases from which she suffered. She is a vigorous, well built child, although of a nervous temperament.

The treatment from the beginning included in addition to approved local measures, a general tonic line of treatment. The condition, however, slowly progressed. Salicylates also failed to arrest the process.

After a careful study of the temperature curve for a week, during which time it was entirely normal, on July 6th a watery extract of tuberculin was administered, the initial dose containing 0.01*mg* of the solid tubercle bacilli. A mild general reaction (temperature 99.6°) followed its use, thus confirming the laboratory diagnosis. Tuberculin was now administered at intervals of three and four days, as conditions

indicated. In ten days improvement was noticeable, the diseased area becoming flatter and more contracted, and the salmon color a trifle paler. This improvement has continued until the present time. Twenty-two injections were given from July 6th to September 21st. The dosage was gradually increased 0.01*mg* at a time, the last dose containing 0.3*mg* of the solid tubercle bacilli. The general supportive and constitutional treatment was continued throughout, including forced rest, fresh air, and hypernutrition. Incidently, one month after the use of tuberculin, the child developed a well marked attack of chorea. Otherwise, her general health improved with the local improvement. From present indications, recovery will be complete. The appearance now is that of a localized episcleritis of a faint brownish tint fading into a pale pink in the periphery.

The case has been one of great interest from a diagnostic, etiologic, and therapeutic point of view.

My suspicions were strongly in favor of a malignant growth until the laboratory report of the removed section gave us the diagnosis of a tuberculous process. Points in favor of malignancy were: first, the report of "papilloma" of the original growth; second, the pigmentation; and third, the rapid appearance of the secondary growth after excision of the first.

Inoculation experiments to reproduce tuberculosis in animals were not thought necessary, and a careful search for tubercle bacilli was not made because the tissue removed was small, and it was thought best to make a serial study of the sections in order to bring out the histologic picture, as the history of the case pointed to malignancy. The laboratory examination, however, the general reaction to tuberculin, and the local results obtained by the use of tuberculin are in themselves *prima facie* evidence of the character of the process. The absence of a local reaction in this case is of no value, either positively or negatively. The initial dose was small—large enough, however, to produce a general reaction without disturbing the local eye condition. I had an opportunity several years ago of observing the administration of too large an initial dose of tuberculin in a case of episcleritis, in which the local reaction was so severe as to require three months of active treatment before the eye showed any signs of improvement. In the ophthalmic use of tuberculin, it is advisable to begin

with the smallest dose that will produce a general reaction without any local disturbance. If, however, a local reaction is urgently demanded, as, for example, in determining the exact character of inflammatory eye conditions in patients known to be suffering from tuberculosis, a sufficiently large initial dose is indicated.

Special interest, however, centres in the etiology of the secondary growth. The possible inoculation from an external source at or after the time of operation may be called into question. Instruments, silk, cotton, and gauzes were sterilized by heat, and all precautions usual in ophthalmic practice were carefully observed in the technique of the operation. Inoculation at the time of the operation, I believe, may be excluded. In the patient's immediate family, so far as I can learn, there is no evidence of tuberculosis. Sanitary surroundings in the home are good and the eye was carefully bandaged for several weeks after operation. Accidental inoculation from without may have occurred, but it is unlikely for the reasons which I have given. It is altogether probable that the contused site of operation furnished a good soil for a growth from an internal focus. Favorable to this conclusion was the constitutional reaction following the use of tuberculin. It is not likely that so small a nidus of tuberculosis would produce any general reaction to tuberculin. I believe, however, it is fair to conclude that this lesion was secondary to an old focus of tuberculosis from within.

A third point of interest centres in the treatment. Under the usual and approved methods of local treatment, together with suitable general regulations, the diseased area grew worse from day to day. When, however, tuberculin was added to our therapeutic measures, improvement was prompt and progressive. The case passed out of my hands and observation before the cure was completed. The treatment, however, is being conducted under my direction, and there was every indication, when I last saw the case, and from subsequent reports, that recovery would be complete.

A coincidence of some interest is the development of chorea during the course of treatment. The child never gave any evidence of pain from the hypodermic needle, and did not seem to dread her visits to my office; but the psychic factor

may have had some determining influence in the production of chorea in an organism which was decidedly neurotic. The tuberculin, *per se*, I believe, did not play a rôle in the etiology.

A FURTHER COMMUNICATION ON MY OCULAR MUSCLE-TUCKING OR SHORTENING OPERATION.

By Dr. GEORGE A. SUFFA, Boston, Mass.

(With six figures in the text.)

SINCE the original article was published in the ARCHIVES OF OPHTHALMOLOGY in May, 1909, I have made several advances in the method of procedure in performing the operation, have added three new instruments, and combined the myometer and clamp. Thus equipped, the operation is more easily performed, and with absolute precision, by means of definite measurements.

The purpose of this paper is to give a full description of the operation as modified by further study and experience and the use of the new instruments; to show how a tenotomized tendon may be gauged and fixed in position and accurate data obtained concerning the extent and position of the tendinous attachment of an ocular muscle, including observations on the important bearing which certain functional and mechanical conditions have upon the operative adjustment of muscular imbalance.

METHOD OF OPERATING IN OUTWARD DEVIATIONS.

After the eye has been prepared for operation and cocaine and adrenalin instilled, the latter to prevent bleeding, a semicircular flap of conjunctiva, convex to the cornea, is laid back, exposing Tenon's capsule and the underlying muscle. A plain tenotomy hook (Fig. 1, F) is passed beneath the tendon close to its attachment to the globe, and Tenon's capsule is cut along both borders of the muscle. The capsule should, however, only be cut to the extent that the muscle is to be

shortened or tucked, as great care should always be exercised to preserve the capsular covering of the muscle and tendon. Measurements of this tendon with the tenometer must be made at this time in the manner to be described later.

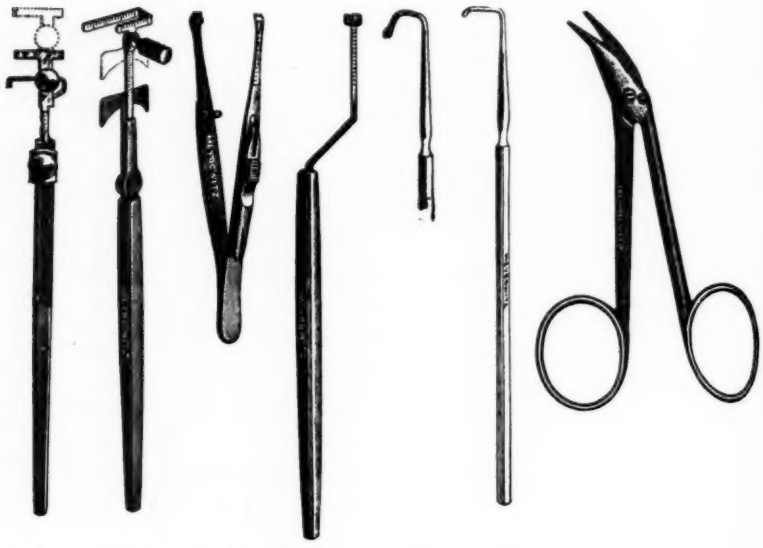

FIG. 1.

(A) Shows the perfected myometer, the clamp being removable;
(B) the tenometer with which the tendon measurements are made;
(C) the graduated forceps to gauge the displacement of a tenotomized tendon;
(D) the graduated regulator to show the distance the tenotomized tendon is displaced while being fixed with the anchor suture;
(E) the retraction hook used to raise the stripped tendon and retract and hold the conjunctival flap in the tucking operation;
(F) the plain tenotomy hook used in cutting the capsule, freeing and stripping the muscle in the tucking operation, in freeing the tendon and cutting the capsule in a tenotomy, and in raising the conjunctival flap when anchoring a tendon in a tenotomy;
(G) the author's angular scissors that cannot buckle when cutting the tendon and with which the tendon can be cut both ways, in the buttonhole operation, without changing the hold.

The myometer (Fig. 1, A) is now placed as shown in Fig. 2, the clamp being fastened one millimetre from the tendinous attachment of the muscle for every four prism degrees of error to be corrected. Extreme care must be taken to hold the hook of the myometer (*a*) parallel with and well into the angle formed by the union of the tendon and the sclera, so that the clamp of the myometer (*b*) will be fastened to the muscle *exactly* parallel to its tendinous attachment, thus preventing torsion of the eyeball when the suture is tied. The suture is then placed as follows: A double-armed No. 6 thread is laid across the muscle between the retraction hook (*r. h.*)

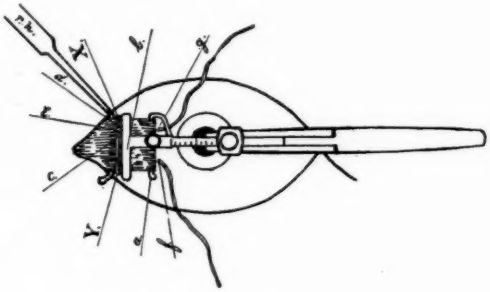

FIG. 2.

and the clamp, at (*c*). The needle attached to the upper end of the suture is then introduced beneath the muscle, including not over one-fourth the width of it, and is made to emerge on the conjunctival surface at (*d*) between the cross-thread (*c*) and the clamp (*b*) close up to the clamp. The needle is then passed over the cross-suture (*c*), is made to pierce the conjunctival and emerge on the scleral surface of the muscle at (*e*) close to the cross-suture.

When drawn taut, the upper loop (*X*) is formed. The lower end of the suture is similarly placed at the lower border of the muscle, forming loop (*Y*) and is drawn sufficiently taut to take up all the slack in the cross-thread and in the loops (*X* and *Y*) but not enough to pucker the muscle. The hook (*a*) is slightly loosened and the needle passed under it, piercing the tendinous attachment and emerging on the conjunctival surface at (*f*) at a distance from the edge of the tendon corre-

sponding to the distance from the edge of the muscle at which the suture penetrated it, *i. e.*, one quarter of its width.

The upper suture is similarly carried through the upper part of the tendinous attachment at (*g*).

When placing the tendinous or holding ends of the suture, the operator should test the needles while they are still in the tissues to see that they have a firm hold, and if not, a deeper hold must be taken, for it is essential that the tendinous or holding ends of the suture be in firm tissues, even if it is necessary to penetrate the sclera superficially.

The needles and the hook of the myometer are now removed, the clamp being left attached to the muscle in order that it may be used later to rotate the eyeball into position when the operator ties the tucking suture.[1]

The external rectus is now tenotomized as follows: The conjunctiva is grasped horizontally over the tendinous attachment with a toothed forceps and incised vertically, the incision extending slightly beyond both borders of the tendon. The conjunctiva is then undermined more or less according to the amount of recession desired.

The tendinous attachment is buttonholed in the middle and a tenotomy hook is introduced into the opening thus made, and pushed through the capsular covering at the upper border of the muscle. A snip with the scissors will aid in bringing the tip of the tenotomy hook through the capsule. The same procedure is followed with the lower border of the muscle. Here, again, the tendon measurements should be made in the manner to be described.

The tenotomy hook is passed under the tendon, including the whole of it, and the capsule cut along both borders of the muscle, the extent of this cutting being governed by the amount of displacement desired. The tenotomy hook is now removed and the tendon grasped at the central buttonhole with the graduated forceps (Fig. 1, C). The remaining attachments of the tendon to the sclera are severed and the recession

[1] In large tuckings, the clamp may interfere with the tenotomy of the externus, in which case it can be loosened and slipped towards, and fastened at, the tendinous attachment, or it can be removed, but if so, it must be replaced later to be used to rotate the eyeball into position when the operator ties the tucking suture.

of the tendon noted on the graduated forceps. This recession should be slightly less than the amount of displacement desired. The writer would suggest that if all tenotomies were done in this way and anchored as follows, there would be no excuse for getting too much displacement of a tendon.

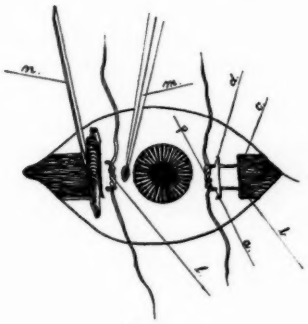

FIG. 3.

The anchor suture (Fig. 3) is now placed. The needle of a single-armed suture (No. 6 thread prepared silk) is made to penetrate the firm tissue of the tendon stump, entering the conjunctival and emerging on the scleral surface one quarter-way in from its lower border at (*a*). The severed tendon is then drawn forward with the graduated forceps (the assistant meanwhile holding the undermined conjunctiva up with the plain tenotomy hook) and the needle passed through the cut end from the conjunctival surface inward at (*b*), in the same relative position that the suture occupies in the tendinous stump. The needle is now passed through the upper part of the freed tendon from the scleral surface outward one quarter way from its border at (*c*) and then through the corresponding part of the tendinous stump from the scleral surface outward, penetrating the conjunctival surface at (*d*). Care should be taken that the tendon ends of the suture are placed in firm tissue before the needle is pushed through. The needle is now removed and the suture laid aside.

On page 257 of the original article, the statement is made that I had not fully determined, in tucking an internus for exotropia, at what time the externus of the same eye should be

tenotomized. I am now satisfied that the tenotomy of the externus should be done before the tucking suture in the internus is tied, that the displacement of the externus should be exactly the same number of millimetres that the internus is shortened, and that the displaced tendon of the externus should be anchored in its new position by a suture until healing has taken place.

A tuck of 6 millimetres has been taken in an internus and a corresponding displacement of 6*mm* made by the writer in the externus for divergent strabismus, according to this rule. It may be necessary to modify this in larger tuckings, making relatively less displacement of the tenotomized external rectus.

The two free ends of the tucking suture are now given a double turn (Fig. 3, *l*). With one hand the assistant seizes the conjunctiva in a firm grasp with the forceps (*m*) vertically between the tendon of the internal rectus and the cornea, or the tendon stump of the externus, if more convenient. With the other hand he holds the shank of the myometer clamp (*n*) and rotates the eyeball into position, enabling the operator to tighten the unopposed tucking suture. The clamp should now be loosened and slipped upward on the loop of the tucked muscle, in order that its under surface may be inspected to see that the base of the tuck is drawn firmly into the angle formed by the union of the tendon and the sclera. Sufficient tension should be exerted to bring these points into close apposition but not enough to constrict the circulation. A final turn in the knot should be taken and it should be square, so that it will not loosen. The conjunctival flap is now replaced and fixed with two sutures. The tucking suture is cut long to protrude between these conjunctival sutures. Before the second conjunctival suture is tied, the muscle loop should be pushed backward and flattened out with a small Stevens tenotomy hook.

The thread used both in tucking and anchoring a muscle should be number 6 to 8 twisted sterilized black silk, steeped in a sterile mixture, consisting of three parts of white beeswax and five parts of white vaseline, according to Worth's method. It is essential to have oiled thread, otherwise it will be difficult to place the tucking suture and to remove it after healing has taken place.

In tucking an externus for inward deviation, the operation is performed in the same way, but under no consideration should the internus be tenotomized. However, the conjunctiva and capsule covering the internal rectus should be buttonholed at and parallel to the upper and lower border of the tendon, and measurements of the tendinous attachments made with the tenometer. At the same time the muscle should be put on the stretch, thus giving the internus its first "breaking-in" and initiating a counter tension, which, with the restraining and recuperative power of the tucked externus, will finally force it to give up its supremacy.

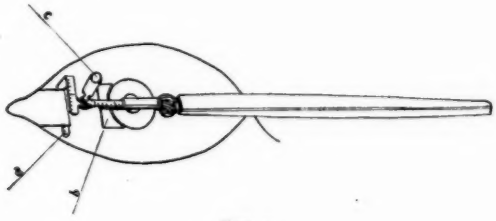

FIG. 4.

METHOD OF MEASURING THE TENDON OF AN OCULAR MUSCLE.

In the tucking operation, as already indicated, measurements of the tendon are to be made after the muscle has been stripped ready for the myometer, and in a tenotomy just before the tendon is severed. In making these measurements, the hook of the tenometer (Fig. 4, *a*) is passed under the muscle and drawn firmly into the angle formed by union of the tendon with the sclera. The concave edge of the slide (*b*) is applied to the corneal margin and the hook is firmly secured with the set screw (*c*) at the angle at which the tendon is attached to the sclera. Care must be taken to have the hook exactly parallel with the tendinous attachment and the curved edge of the slide exactly over the sclero-corneal margin. The width of the tendon should be noted on the graduated hook.

The instrument may now be removed and laid aside, and the readings taken at the convenience of the operator, unless the opposing muscle is to be dealt with, in which case the

readings must, of course, be recorded before measurement is made of the opposing muscle.[1]

I think it will be generally admitted that the scientific way to correct esotropia is by shortening the external rectus muscle of the converging eye (in the higher degrees, of both eyes) while still preserving the integrity of the tendinous attachment of both lateral muscles, and that relatively few operators are taking this method, but that by far the greater number tenotomize the internus, although realizing it to be a faulty operation. The reason for thus adhering to tenotomy is to be found in a very natural uncertainty as to results, fostered by the numerous vague and indefinite methods of tucking or advancing and their multiplicity of modifications, rather than in the intricacy of the procedures as compared with a tenotomy.

After tenotomy of an internal rectus muscle, especially a large tenotomy, there inevitably must be curtailment of muscular action in the muscle involved, which precludes all future possibility of obtaining normal binocular movements, as long as that muscle remains displaced. There is a positive and absolute loss of converging power, a power needed at all times, but especially necessary as age advances, when that function normally becomes weakened—a condition still further aggravated by the adoption of presbyopic lenses.

I have observed that the tendinous end of the externus of the highly esotropic eye has become markedly attenuated, the muscle appearing more like a connective-tissue band than a muscle. This thinning is, in all probability, due to the long-continued tension of the internus holding the eye in constant convergence, thus stretching the externus windlass-wise over the globe.

A study of the accompanying diagrams of the orbital cavities and the position of the eyes within these cavities will reveal the mechanical reason for the positive and absolute

[1] These tendon measurements have no immediate bearing upon the operation as at present performed, but are taken for the purpose of accumulating a fund of accurate data concerning the tendinous attachments of the ocular muscles which will be of value in deciding how much influence variations of angle, position, width, etc., exercise upon muscular imbalance and how far they are to be taken into account in its correction by operative means.

loss in action of a tenotomized internal rectus muscle, and show that this loss of action cannot be recovered unless the tenotomized muscle is brought back to its original attachment. It also shows that a tuck in the externus not only reinforces the muscle, aiding it to recuperate and regain its normal restraining influence, but also re-establishes the normal contact arc of the internus, while retaining the original tendon attachments of both muscles.

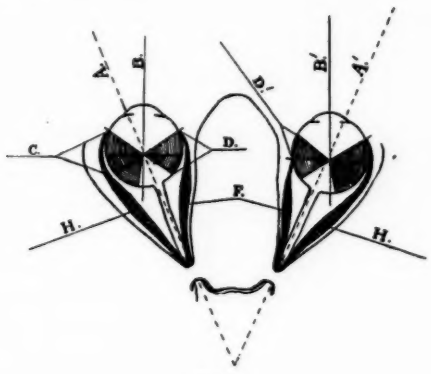

FIG. 5.

A A^1 = Orbital axes.
B B^1 = Visual lines.
F = Internal recti.
H H = External recti.
D = Normal attachment of left internal rectus, showing fourteen millimetres of movement and seven millimetres of contact arc.
D^1 = Seven millimetres' displacement of right internal rectus (by tenotomy), with seven millimetres of mechanical loss of movement and a complete loss of the contact arc.
C = Normal attachment of left external rectus, with possible mechanical action of twenty-one millimetres and a contact arc of fourteen millimetres.

The orbital outlines in figures No. 5 and No. 6 were traced from a skull cut in the median horizontal orbital plane, terminating just at the upper part of the optic foramen. The dimensions and position of the ocular structures were arbitrarily fixed, the diameter of the eyeball being considered 24

millimetres, the centre of rotation 10 millimetres from the posterior surface, the attachment of the interni 6½ millimetres from the cornea, and of the externi, 7 millimetres, all dimensions, attachments, etc., corresponding with the average obtained from data at hand.

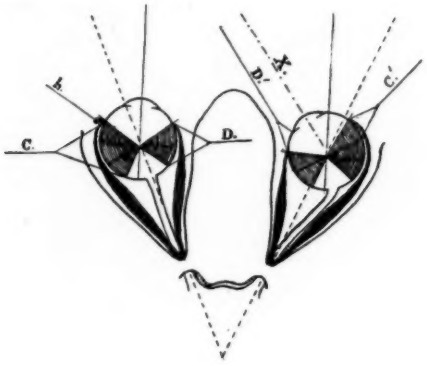

FIG. 6.

D^1 = Normal attachment of right internal rectus, showing convergent strabismus of seven millimetres, with a consequent contraction of the muscle of seven millimetres and a complete loss of the contact arc.

C^1 = External rectus of right eye stretched seven millimetres, increasing the contact arc seven millimetres.

X = Displaced visual axis of right eye.

C = Shows the author's tuck in a stretched left external rectus muscle, correcting seven millimetres of convergent strabismus, bringing the eyes into parallelism, retaining the original tendinous attachments, re-establishing the normal contact arc of the internus and normal relations between the muscles of the two eyes, making it possible for them to perform normal binocular movements in all positions.

A full realization of the facts here shown and their relation to tenotomy of the internal rectus, proving it to be an absolutely faulty operation, forever destroying the possibility of normal binocular muscular movements, together with the uncertainty and vagueness surrounding the various methods of tucking and advancing ocular muscles, supplied the incen-

tive which caused me to attempt to work out a scientific method that would accurately perform and measure the operative procedures, hold the operated muscle or muscles exactly as placed until healed, preserve the original tendinous attachments and integrity of the muscles, in inward deviations, and at the same time afford accurate data of the tendinous attachments of the muscles concerned.

The rule of one millimetre of correction for every four degrees of prism deviation appears to hold good in *outward deviations*. Adjustments of from 10 to 12 millimetres can, and should be made on the deviating eye,—which means a correction of from 40 to 48 prism degrees of error. Should the error be greater, further correction must be made on the other eye. Cases requiring further operation should be held under observation for at least six months and repeated tests of the muscular condition made before the second eye is operated upon. The data thus obtained should be used as a guide in the final operation.

N. B.—See page 250 for statement on the modification of this rule that may be necessary in the high degrees of error.

All the corrections for esotropia, so far made, have been according to the rule of one millimetre for every four prism degrees of error and have varied from 5½ to 12*mm*, most of them having been of too high degree to be corrected by operation upon the deviating eye only, the deviating eye usually being amblyopic as well as highly hyperopic.

Whether partly or fully corrected, during the first two or three weeks after the operation there has been spasmodic convergence (of short duration usually) of the operated eye, apparently to the full degree of the original error, followed by periods of relaxation to slightly less than the correction made by the tucking. This spasmodic contraction of the internus finally ceases and there is a progressive return to the correction made. In other words, for a period varying from six months to three years, there has been a progressive decrease in the remaining convergence or even a tendency to exophoria in the cases fully corrected according to the rule given.

The occurrence of this uniform increased action after taking the slack out of the stretched externus shows that this muscle

possesses a remarkable recuperative power. Just how much this recuperative power of the externus and the normal weakening of the contracted internus will have to be reckoned with in these cases, time and careful observation of many operated cases must show. From the limited number that I have been able to follow up carefully, no definite statement can be made. At present I can only sound a warning note by saying that one should wait at least a year before correcting the remaining convergence by tucking the external rectus of the other eye, meanwhile using all available means to relax the contracted internus. This recuperative power of the externus and relaxation of the internus absolutely prohibit tenotomy of an internus at any time.

A detailed report of operated cases will be given at a later date, as it would prolong this paper unduly.

By obtaining scientific data from accurate measurements as described, by keeping cases under observation during the healing process, and by repeated carefully-made and recorded measurements of the muscle status, for a long time after the operation, in a large number of cases, we may hope to evolve a definite rule for the operative adjustment of ocular imbalance, in case the one given in this paper fails to hold good.

I earnestly appeal to operators to adopt, if not mine, at least some method that observes the essentials of accurate data and positive adjustment, so that operative procedures upon the eye muscles may be removed from the uncertainty which at present involves them, to be established at no distant day upon the definite mathematical basis which alone will insure uniform results.

SUMMARY

I. *In tucking an ocular muscle:*

(1) Care should be exercised to preserve its capsular covering.

(2) The angle of the tendinous insertion of the muscle should be noted and the tucking suture put in parallel to it by means of the myometer so that there shall be no torsion when the suture is tied.

(3) The holding ends of the suture should be firmly anchored in the tissues.
(4) One millimetre of tucking is to be done for each four prism degrees of error.
(5) The suture should be tied with consideration for the circulation and with a square knot.

II. *For the correction of outward deviations:*

(1) The external rectus should be tenotomized before the tucking suture in the internus is tied.
(2) The displacements of the externus should be exactly the same number of millimetres that the internus is shortened. (Note possible modification, page 250.)
(3) The displaced tendon of the externus should be anchored in its new position by a suture until healing has taken place.

III. *For the correction of inward deviations:*

(1) The internal rectus is not to be tenotomized under any circumstances.
(2) In order to aid the tucked externus and give the internus a "breaking-in," the internus should be stretched.

IV. *Measurements of the ocular tendons with the tenometer for the purpose of getting accurate data:*

(1) In tucking operations measurements are to be made after the muscle has been stripped ready for the myometer.
(2) In tenotomy just before the tendon is severed.

V. *Diagrammatic illustrations of:*

(1) Mechanical disadvantages of the internal recti muscles.
(2) Mechanical advantages of the external recti muscles.

VI. *The external rectus has remarkable recuperative powers, while the internal rectus, per contra, relaxes as shown by observations of operated cases.*

VII. *Operators should co-operate in getting data in order to establish definite rules for operating on the ocular muscles.*

REMARKS ON SOME OF THE RECENT ESSAYS CONCERNING THE PATHOLOGY OF THE LENS

By Professor C. HESS, Würzburg.

Translated from the *Archiv f. Augenheilkunde*, 1911, No. 1, by Matthias Lanckton Foster, M.D.

I SAID, in connection with the report of experiments concerning lightning cataract in 1888, "that the lenticular opacity following the electric stroke is for the most part, though by no means exclusively, a consequence of the death of larger or smaller groups of capsular epithelia." "Whether and in how far the lenticular fibres themselves are affected directly by the lightning, is difficult to determine." I did not deny the possibility that perhaps a portion of the fibres were injured so that they might die. I said later that the lenticular opacity "is in reality the consequence of the death of a larger or smaller group of living lens cells." (I avoided the expression lenticular epithelial cells.) In speaking of naphthalin cataract, I assented to the theory that in consequence of the overfeeding with naphthalin, substances exert a toxic action "upon the living elements of the lens, primarily the capsular epithelium." In speaking of senile cataract, I finally wrote: "I have occasionally noticed in my studies concerning lightning cataract that it is difficult to differentiate in how far the fibres were themselves directly injured by the lightning; likewise I could not be certain whether any destruction of cortex was wholly the result of the destruction of the epithelium, or due in part perhaps to the direct action of the injury that killed the epithelium upon the cortical fibres themselves. We must not forget that a sharp differentiation of the lenticular elements into epithelia and fibres cannot be made, especially in the equatorial portion; for in the young cortical fibres we meet with living, growing,

nucleated cells which the more resemble the epithelial cells, not only anatomically but in their biological properties, the younger they are. Hence it is understandable that an early and extensive extinction of fibres is found in this region in senile cataract, the same as in naphthalin cataract."

This shows that I have emphasized in many places that not only the capsular epithelium, but the lenticular fibres as well, must be taken into account in the injuries which the lens suffers, that lead to opacity.

It is therefore surprising that Kuwabara should credit me with a "capsular epithelium theory," which he then overthrows to set up "his theory," that "the direct injury of the lenticular fibres is due to the same cause as injured the capsular epithelium."

He writes among other things: "I have shown in my work concerning lightning cataract that the lens substance itself is injured by the lightning and nothing can be said of a nutritive disturbance caused by an injury of the capsular epithelium."

In this article, which confirms my former findings, Kuwabara writes that I had assumed the death of the lenticular epithelial cells, while I had spoken of the lens cells and the possibility of a simultaneous injury of the fibres together with the epithelium. He has overlooked all the places in my works in which I discussed the involvement of the lens fibres in the disease that leads to cataract. As the rest of his views are based on this erroneous assumption I desist from their consideration.

Possek advanced in 1909 the following theory concerning the etiology of cataract: "Such nutritive disturbances of the lens may be caused by the nutritive material for the lens being affected in the blood serum so that it is either destroyed or undergoes some other change so it cannot reach its destination. There is, in my opinion, a deficit of specific nutritive material in the nutritive fluid of the lens undergoing cataractous change."

With this hypothesis Possek advances anew an idea to which I have repeatedly given expression as one of several possibilities. In an article on lenticular opacities and their relations to general diseases I stated for the first time (1896) that

we can certainly imagine that the nutritive fluid of the lens may be quantitatively and in many cases qualitatively changed in certain forms of cataract, among which I named senile cataract. Later (1905) I said: "It may be that certain elements necessary for the preservation of the vitality of the cells are wanting in the nutritive fluid surrounding the aging lens, or are present in insufficient quantity."

During the description of the pathological lens of an embryonic chicken, in which there was a disturbance in the constriction off of the lens vesicle, I indicated the possibility that lamellar cataract might have a similar etiology, in the following words: "It might be that if the development of the animal had not been interrupted in this case, the lens vesicle would have become constricted off, after the mechanical obstruction to the closure had been removed by the destruction of the proliferated fibres. If normal lenticular fibres had then developed, the picture of a congenital central cataract would essentially have been produced. The close relations, both clinically and anatomically, between lamellar and nuclear cataract give rise to the thought that the etiology of the former may be similar."

E. von Hippel objects to these reflections; he says: "The constriction off of the lens normally takes place in the fourth or fifth week. The lenticular fibres which are formed prior to the constriction off are, according to Hess, abnormal; those that form later are normal." The latter statement is not correct. I have not claimed that the fibres after the constriction off are normal. I simply said that if normal fibres had developed in the case described after the closure, the picture of a central cataract would have been produced. How little I have held the theory ascribed to me by von Hippel is shown, among other things, by the fact that I discussed the possibility of a similar etiology of a case of congenital total cataract with the following words: "It is suggested by what has been said that this congenital total cataract may be considered a far advanced lamellar cataract. We must then suppose that the consequences of the pathological disturbance which produced the changes of the nucleus and of the perinuclear layers characteristic of lamellar cataract, acted also in that period in which the cortical layers of the lens were being established.

While these develop normally in the usual forms of lamellar cataract, they were destroyed in this case."

When von Hippel writes, "The size of the zone of lamellar cataract can thus be brought into harmony with Hess's hypothesis only by the assumption that the newly formed fibres remain abnormal in their development for several months after the constriction off of the lens vesicle," such a theory harmonizes perfectly with views enunciated by me.

I have formerly shown that my hypothesis for the etiology of lamellar cataract furnishes a satisfactory and uniform explanation for several conditions that are otherwise difficult to understand. Two of these are the displacement of the opaque nucleus into the neighborhood of the posterior pole, which I have demonstrated repeatedly, and the comparatively frequent occurrence of a distinctly visible anterior polar opacity in lamellar cataract.

I have repeatedly and emphatically pointed out the importance of heredity in this form of cataract; a number of years ago I made the suggestion "to try to solve the question of the genesis of this form of cataract by systematic breeding."

In the above mentioned article, "Concerning Lenticular Opacities and their Relations to General Diseases," I classed senile cataract among the constitutional forms. I pointed out that pathological changes in the entire mass of fluid in the organism may give rise to more or less extensive opacities of the lens, and we can certainly imagine that in such forms of cataract "the nutritive fluid of the lens is changed qualitatively (in many cases quantitatively also), that the vitality of the lenticular cells and fibres is impaired thereby and consequently the fibres become opaque and destroyed." Later I expressed the same views in the Graefe-Saemisch *Handbook*.

Elschnig (1911) opens an attack on my essay in the *Handbook* with the doubly strange remark that I have been influenced by the biological researches to which I am accustomed in my institute. His theory suffers by the proof that my former researches are not known to him.

Elschnig further says that he has established "the regular involvement of the nucleus in the development of senile cataract," and writes that he can "in no way accept the view

concerning the late and rare involvement of the nucleus in the development of senile cataract." For this error also Elschnig finds no ground in my article. He examines extracted cataractous lenses in a "white porcelain cup filled with physiological salt solution, on the bottom of which is a small black, glass plate, with a millimetre scale engraved on it," brings "the lens by means of a cataract spoon alternately over the white bottom of the cup and the black plate," and thinks that such an observation of the surface of the extracted lens permits a perfectly satisfactory establishment of all the conditions to be taken into account. He does not divide the lenses and examine their cut surfaces, as I am accustomed to do; he easily convinces himself in the way described that the pathological changes in the extracted lenses, which during life appeared as subcapsular cortical cataracts in which the entire cortex was opaque, as a rule extend to the outermost portions of the nucleus lying next the cortex, while the nucleus itself may appear more or less normal, as is generally understood. I have particularly mentioned and illustrated that we may find the periphery of the nucleus permeated by numerous little round spaces, in such cortical cataracts, which I consider the commencement of the destruction of the parts of the nucleus affected. Elschnig's statement, "that the total cataract with a brown nucleus occurs commonly in older, that with a gray or white nucleus, in younger, individuals," agrees with the experience of us all, that in general the lenticular nucleus opposes more and more resistance with increasing age to the destruction beginning in the cortex.

Elschnig approves of my idea of the first demonstrable opacity in the forms of cataract now under consideration, with the words: "It is also to be considered the rule, from the prolonged observation of patients with cataract from its commencement until the operation, that the subcapsular opacities of the cortex, which almost always begin in the posterior cortex near the equator, have added to them in their further advance the opacity of the nucleus." When Elschnig continues, "it often cannot be determined whether both appeared simultaneously," it can certainly be understood that the question as to the beginning of the senile cataract cannot be taken into account in cases that are so far advanced that

the starting place of the opacity can no longer be made out.

Elschnig also writes: "The senile cataract is as a rule a total cataract, *i. e.*, the nucleus and cortex are opaque, and deviations from this rule are comparatively rare exceptions." This is in contradiction to the generally accepted fact that most subcapsular cortical cataracts are not total cataracts, but are opacities that begin and continue for a greater or less length of time to be confined to the cortex, without becoming total; he contradicts also his own words, quoted above, in which he adds the opacity of the nucleus to the opacities beginning in the cortex "during their further advance."

Elschnig closes with the words: "It scarcely needs to be emphasized that the establishment of the regular involvement of the nucleus in the development of senile cataract does not support Hess's theory of the etiology of senile cataract." It is not very clear what this generally known fact of the involvement of the nucleus, which was discussed by me, but which Elschnig thinks he was the first to establish, has to do with my conception of the origin of senile cataract; he agrees in all essential points with my account of the beginning of subcapsular cortical cataract and the subsequent involvement of the nucleus. His error is chiefly due to the fact that he contented himself with the contemplation of extracted cataracts, which were generally fairly far advanced, while my account of the development and course of the lenticular opacities was based on the observation of all cataracts, especially on a large number of commencing forms, and was not content with the macroscopical appearance of the surface, but studied macroscopically and microscopically the cut surfaces of the nuclei.

Elschnig agrees on the whole with my subdivision of cataracts, in accordance with the starting points of the opacity, into subcapsular, supranuclear, and intranuclear or nuclear, but unites the first two as cortical cataracts and correspondingly speaks of subcapsular and supranuclear cortical cataracts. I do not consider this expedient, because the supranuclear cataract differs essentially in its etiology, both clinically and anatomically, from the subcapsular, and this difference is effaced without cause if it is grouped with the form in which the opacity commences just beneath the capsule under the

common term "cortical" cataract. There are no advantages of the name suggested by Elschnig to counterbalance this essential disadvantage.

Elschnig raises philological objections to my term "intranuclear" cataract, and writes: "intra means within, intranuclear cataract is therefore a cataract within the nucleus of the lens." Aside from the fact that my term could be misunderstood by nobody, I have ascertained from philological authorities that it is unobjectionable and has a single meaning. I avoided the term nuclear cataract, adopted by Elschnig, because it has been applied to several other kinds of lenticular diseases.

In addition to the three forms into which I divided cataract, Elschnig proposes: 4, the peculiar senile cataract, or senile total cataract, with its regressive metamorphoses; and 5, the cataracta brunescens sive nigra. In regard to the latter I have formerly stated my position and Elschnig advances no new point of view. In regard to the former I think it questionable whether the name "cataracta totalis incipiens" will be adopted because of the contradiction contained in itself. No cataract begins in all parts of the lens at once and when it is incipient it is not total; when it becomes total it ceases to be incipient. When an incipient becomes a total cataract, whether after months, years, or decades, we cannot tell with our present means; in this Elschnig agrees with me.

A SUCCESSFUL METHOD OF CORNEAL TRANSPLANTATION.

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(*With one Appended Plate and three figures in the text.*)

IN a few years a century will have passed since the first attempt at corneal transplantation was made by Riesinger, on animals. This idea was followed up with enthusiastic hopes, as is evidenced by the numerous papers of the succeeding decade, which, in spite of repeated failures, took up the question again and again. And even after the keratoplastic attempts fell into discredit for a long time, on account of the almost constant totally unfavorable results which followed the transplantation of the cornea of animals, and the bizarre idea of implanting an artificial cornea, there appeared, in the beginning of the 70's, another enthusiastic supporter in the person of A. von Hippel, who, through his untiring efforts, gave an impetus to the universal reconsideration of the question.

Although, in this second epoch, the therapeutic results were very unsatisfactory, yet we owe to this period our clearer conception of the possibility of, and the favorable requisites for, transplantation of corneal tissue.

It is unnecessary to go into these papers here, as they have all been reviewed in Marchand's *Prozess der Wundheilung* (1901, Chapter 31), and in Salzer's "Kritischen und literarischen Studien ueber Transplantation in Hinblick auf die Frage der Keratoplastik" (1900, *Zeitschr. f. Augenheilk.*, part 3,

p. 516). It will be sufficient for me to make a short résumé of the stand taken in regard to the question at that time, in order to deduce from it my own proposition, which, judging from the results obtained up to the present in animals and man, promises to be of practical utility.

It has been attempted to solve this question in three ways. The first method, namely, to replace a portion of the opaque cornea by inanimate transparent material, may be considered to have been definitely abandoned. Such artificial corneæ were first employed by Nussbaum in the form of a small glass window inserted into the opaque cornea (1856). V. Hippel was influenced to attempt his plastic work on the cornea (1877), by the failure of similar experiments with a removable pane of glass inserted into a gold frame, which he fitted into an opening in the cornea. The results which Dimmer reported at the Heidelberg congress in 1889 were no better, namely, inserting an almost transparent piece of celluloid into a trephine opening in the cornea. The last attempts of this nature were made by Salzer (1898, 1900), who attempted to give the prothesis greater security, by attaching it with several small hooks. In one case, the prothesis held for 2¾ yrs., and in another case which held for 8 months, the optical effect was a very good one.

These attempts do not promise very much, as in the majority of cases, an opaque membrane forms behind the prothesis, or the prothesis falls out, either as the result of a genuine foreign body expulsion, or (as Salzer thinks) as the result of intraocular pressure and closure of the lids.

Whether the recently reported attempts of Salzer to transplant preserved pieces of the cornea of one animal into the cornea of another animal of different species will, in addition to their great theoretical interest, have any practical significance, it is too early to judge.

The method which was at first exclusively employed was the so-called total keratoplastic. It consists in the removal of the total thickness of a portion or all of the opaque cornea, and replacing it by a piece of normal cornea of the same size (Himly, Riesinger, 1818).

From the numerous attempts made in this direction—namely, since the improvement of the method by the use of

v. Hippel's trephine—the following conclusions may be drawn:

A piece of a guinea-pig's cornea, removed by means of a trephine and immediately reimplanted, can heal in and, after an initial clouding and vascularization, may become completely transparent again (compare Marchand, Wagenmann, etc.).

There is much less likelihood of the flap healing in and remaining transparent, in transplantation between animals of the same or different species.

The therapeutic application of the method to the leukomatous human eye has, as yet, as far as the optical effect is concerned, been successful in only a few exceptional cases. The transplanted corneal flap heals in, in the majority of cases, but the contact with the aqueous, the injury of the endothelium and Descemet's membrane, the cicatricial character of the surrounding corneal tissue, and the formation of anterior synechiæ, causes, in almost every case, a complete opacity of the flap, as the result of the emigration of cells and vascularization of the flap, often accompanied by the formation of a membrane behind it. Although the optical effect of total keratoplastic (especially in leucoma adherens) is seldom good, yet it has been proven efficacious in guarding against the formation of staphyloma, in leukomatous corneæ (v. Hippel, Fuchs), a standpoint which does not concern us here.

In order to overcome the grave dangers attendant upon the opening of the globe in total keratoplastic, there was soon devised a plan, whereby only a portion of the thickness of the cornea was removed, Descemet's membrane being allowed to remain. Following the direction of Walters, this partial keratoplastic was first performed by Koenigshoefer (1839), Muehlbauer (1840), and Hauenstein (1843), who, in their experiments by this method, with the cornea of animals, seem to have had good results in a portion of their cases,—even as regards the preservation of transparency.

The first complete reports on partial keratoplastic were published by Duerr (1877–1881), and v. Hippel, who was the first to use a trephine for this purpose.

Duerr, believing good nutrition of the transplanted flap to be of prime importance for successful healing, transplanted

a superficial corneal flap together with an adjacent conjunctival flap, with good success, a number of times in guinea-pigs and man. In every case, there was, in the first weeks, intense opacity with vascularization of the flap from the limbus. Then there followed a gradual disappearance of the vessels, with almost complete transparency, this transparency remaining for a considerable time in some cases. However, in all except one case, the transparency did not last.

The next one to perform partial keratoplastic was A. v. Hippel (1883). With the aid of the trephine, he transplanted superficial corneal flaps from animals to man. The flaps became completely opaque. V. Hippel attributed his failures to traumatism to the partial corneal flap. In order to avoid this traumatism, he used as a flap the entire thickness of the cornea of a guinea-pig. In these cases, the flap healed in well, with preservation of a fair amount of transparency. The cases reported by others in the succeeding years, which were done by von Hippel's partial keratoplastic, were not very satisfactory. The difficulty of the method lies largely in the technique.

According to Salzer, the value of partial keratoplastic may be summed up as follows: "In partial keratoplastic, undertaken on the clear cornea of animals, or on the superficially opaque human cornea, the flaps may remain permanently clear. However, even in partial keratoplastic, the flap often becomes opaque as a result of the growing in of vessels and cells."

It seemed to me that the most important consideration in the province of corneal transplantation was to improve the operative technique in such a manner as to furnish more favorable conditions for the healing in of partial corneal flaps.

Previous experiences have taught us the following facts:

1. Partial keratoplastic promises the best optical results in corneal opacities due to parenchymatous keratitis, corneal ulcers, lime-burns, etc., in which optical iridectomy can do no good. Extensive anterior synechiæ are unfavorable factors, as they result in the rapid entrance of numerous blood-vessels into the transplanted tissue, thus favoring its permanent opacity.

2. Human corneal tissue is the most favorable material for the formation of the flap, and it is not impossible that the

bad results after the method of Duerr and von Hippel were partly due to the use of the corneæ of animals. That autoplastic operations offer the best chances in this domain, also, is proven by the successful case of Plange (1908), who was placed in the unusually fortunate position of utilizing a portion of corneal tissue from the other eye of the same individual for the formation of his flap.

3. In regard to the gaining of the flap, it has been repeatedly emphasized on all sides, that there should be perfect conformity in shape and size between the flap and the defect. In order to overcome this problem, there have been devised the various modifications of the trephine. On the whole, however, one gets the impression that the importance of this consideration has been widely overestimated. For, in the first place, the desired effect is not entirely achieved with the trephine, on account of the shrinking of the flap, after it is cut, and secondly, those operators who cut their flaps about the size and shape of the defect (Hauenstein, Duerr, Plange) had just as good results as the adherents to the trephine.

4. Very important, on the other hand, is a consideration, which was particularly emphasized by v. Hippel, namely, the greatest delicacy in handling the flap. It should be touched by as few instruments as possible. For this reason, partial corneal flaps obtained with the trephine cannot be used. Total corneal flaps can be obtained in this way with much less traumatism. Furthermore, the flaps should not be allowed to come in contact with coagulating or antiseptic fluids. If sufficient care is not taken, there are likely to occur local patches of necrosis in the flap, which later give rise to the formation of blood-vessels and cells, thus resulting in loss of transparency.

5. The condition of the surface of the defect upon which the flap is to lie is of some importance. It should be fairly smooth, which is especially difficult in using the trephine. Plange's method of shaving the cornea layer by layer gives a fairly smooth surface. Hemorrhages from vessels in the floor of the defect are disturbing factors, especially in those methods where there is no fixation of the flap. In such cases, it may be washed away by the blood.

6. Various methods have been devised for fixing the trans-

planted flap. Corneal sutures have been largely given up, as they give rise to local necrosis and infiltration. Zirm fixed two sutures into the conjunctiva, allowing them to cross in front of the cornea. This method may give rise to pressure necrosis of the flap. Plange's method of implanting the flap with its margin into corneal pockets is very difficult to carry out. Others have drawn conjunctival flaps over the corneal flap, but without good results. In short, this important question has not yet been definitely solved.

7. There has also been a great deal of discussion in regard to the significance of the nutrition of the transplanted tissue. On the one hand, some have laid great stress upon the rapid inclusion of the implanted tissue into the sphere of the tissue metabolism, and upon the importance of obtaining the best nutrition of the flap in various ways. Here belongs Duerr's method of transplanting a corneal flap with a small adjacent conjunctival flap, suturing the conjunctiva at the limbus. For the same reason, many prefer to implant the flap into vascular scar tissue, thus guaranteeing rapid nutrition of the flap. For the same reason, Zirm and others have advised drawing conjunctiva over the scarified corneal periphery. Salzer, however, on the strength of his experimental researches, doubts the expediency of this procedure.

On the other hand, many authors believe that an abundant vascular supply is the cause of failure in many cases. Salzer claims that "the more vascular the surroundings, the more certainly will the flap become opaque." This view is not without reason, as it has often been observed that in cases of transplantation in a vascular cornea, the healing is very prompt, but there is a very rich growth of vessels into the substance of the flap, resulting in the formation of new connective tissue, with more or less permanent opacity of the cornea.

As the result of the experiences of others, and my own experiments, I believe, contrary to Salzer, that it is very important to have the best possible nourishment for the transplanted flap, so as to avoid necrotic changes in the flap, with the resultant reaction. But I do not believe that the proper method to attain this end is through vessels in the floor of the wound and the edges of the flap. The problem seems to be, to make the nutritional conditions of the flap as nearly as possible like

those in which it existed before. Thus I agree with Duerr and Plange, who say that the large wound surface undoubtedly provides the flap with relatively favorable nutritional conditions, and, when the removal of the opaque layers has been complete, relatively normal ones.

PERSONAL ATTEMPTS.

The impulse toward my experimental work in partial keratoplastic was furnished by the observation of two cases of injury of the cornea in the eye clinic at Giessen. In both cases, there was a wound of the cornea, in the shape of a triangular flap, without infection. In one case, the base of the flap was at the limbus, and in the other, it was centrally situated. In the first case, there was rapid healing with preservation of the transparency, and in the second case it resulted in opacity. This clinical difference was due, undoubtedly, to the more favorable nutritional conditions, with peripheral position of the base of the flap.

The conclusion seemed natural, that in partial transplantation of corneal tissue onto an artificially produced defect, similar differences in nutritionm ust be factors, and that transplantation with the trephine, especially when it is centrally placed, offers relatively unfavorable chances for success. I concluded, therefore, without knowing that Duerr had aiready tried a similar plan, to make keratoplastic attempts of such a nature that a portion of the thickness of the cornea with an attached portion of the conjunctiva was transplanted into a corresponding defect. The failure of the first attempt in a guinea-pig, on account of insufficient fixation of the flap, led to the working out of a method which has proven successful in the human eye, and which I would like to make public, in order that it may be tried by others.

In my experiments with guinea-pigs, I removed a corneal flap from each of two animals, and transplanted the flap of one animal on the cornea of the other. The conjunctival sac was cocainized and washed out with normal salt solution. The flap was rectangular, and reached from the top to the bottom of the cornea. In order that the width should be the same all over, I had constructed a fork-shaped instrument, on the end

of each of whose tines is attached a small semicircular blade, with a radius of about ¾*mm*. Fig. 1 shows this simple instrument, which I had made of varying widths (4–6*mm*), in order to cut flaps of varying widths, and with varying radii of the blades, to meet the varying thicknesses of the cornea. The double knife, Fig. 1, is applied to the upper margin of the cornea, and carried vertically downward to the lower limbus, thus obtaining two vertical parallel incisions in the cornea, which do not perforate it. These incisions form the lateral limits of the flap. At the upper limit of the flap, a corresponding portion of the conjunctiva is dissected away. This conjunctival

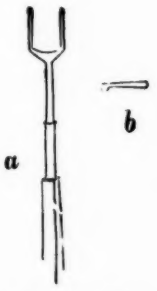

FIG. 1.—*a*. Double knife seen from above. *b*. Lateral view showing a single prong with semicircular cutting end.

flap is seized with forceps in the left hand, and with a narrow knife the sclera is incised at the limbus, without opening the anterior chamber, as shown in Fig. 2. The knife then passes into the cornea, more or less deeply, depending upon the depth of the opacity. The forceps now grasps the more resisting scleral margin, and the knife passes through the cornea with a sawing motion, in a tangential position, until it reaches the lower limbus. This step of the operation—even in the very convex cornea of the guinea-pig—is much easier than is to be expected, as the knife seems to remain in the same plane, between the corneal lamellæ, without any difficulty. However, the marked bulging of the guinea-pig's cornea makes it difficult to keep the incision exactly within the lateral limitations of the flap as marked out by the two parallel incisions. However, as the flap becomes firmly fixed by sutures, this

does not seem to matter much. When the knife reaches the lower limbus, there is made a small conjunctival flap with a rim of sclera attached, just as in doing a cataract operation. The flap is grasped by both conjunctival flaps, and immediately placed over the corneal defect of the other animal. If the wound-surface is covered with blood, it may be carefully washed off with salt solution. With some practice, the removal and transplantation may be done so rapidly that there is scarcely any or no primary haziness of the flap. Two

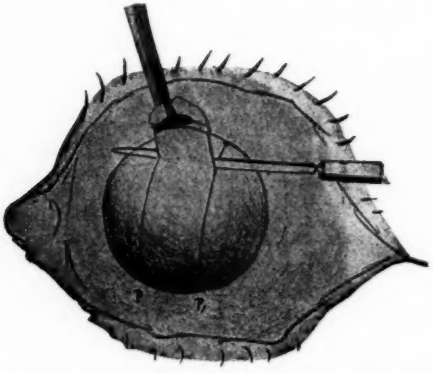

FIG. 2.—Removal of rectangular corneal flap with a knife. p and p_1 indicate limiting lines made with the double knife.

sutures are passed through each of the conjunctival flaps at the limbus. This fixation of the flap, especially when the scleral tissue is utilized, is perfect. It is advisable not to leave the sutures in too long, in the guinea-pig, as they may give rise to profuse secretion and even infection.

The most important consideration of this operation is in the provision for good nutrition of the transplanted tissue. This is assured by the broad surface of contact. The more complete the removal of the opaque and vascularized lamellæ has been, the better will the nutrition of the flap be. Especially valuable for the nutrition of the transplanted flap is the presence of the scleral and conjunctival tissue in the ends of the flap. As we know from the cataract operation, union between the conjunctival flap and the surrounding conjunctiva

occurs very quickly, so that circulatory changes soon take place in a normal manner in the transplanted flap. Even in those operations on guinea-pig's eyes, where secondary infection occurred, the loss of tissue occurred first always in the centre of the flap, whereas the portions near the limbus, owing to their better nutrition, remained intact.

Another important consideration is the question of fixation of the flap. The methods heretofore in use have not been satisfactory. Especially in v. Hippel's partial keratoplastic,

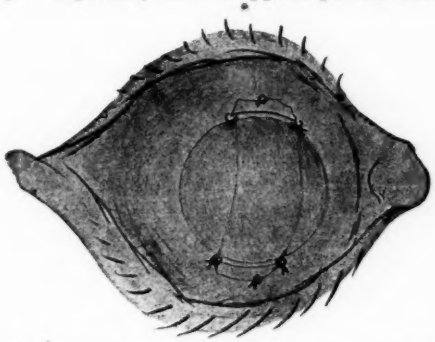

FIG. 3.—The transferred fixed corneal flap with overhanging conjunctiva.

there is a repeated complaint that the flap is displaced either by bleeding or through winking. The four conjunctival sutures in the above described method hold the flap in place very firmly. In the guinea-pig, the nictitating membrane sometimes undermined the edge of the flap. In this way several of my attempts on guinea-pigs failed. Yet in spite of this mishap the entire flap was not displaced, as the flap rapidly united at the limbus, and only became undermined at the centre. In the guinea-pig, this accident could be avoided by removing the nictitating membrane, or by placing the flap horizontally, but in the human eye the vertical position is the most favorable, as this position minimizes the bad effect of the lids in winking.

It is especially important that this fixation should occur without injuring the transplanted corneal tissue by the sutures. As far as the conformity between flap and defect is concerned, this is assured by the parallel vertical incisions made with the forked knife.

Another advantage of the method is in the size of the flap. It is not always possible to estimate the depth of the opacity in the cornea. It usually varies in depth in different parts of the cornea. The larger the denuded area, the more likely is it to include a clear portion of the remaining corneal tissue. If this clear portion lies peripherally, it can be utilized by an optical iridectomy, done later. In addition to this, we must take into consideration the fact that in transplantations we have not ideal healing conditions. Localized opacities, as the result of vascularization, are the rule. Consequently, the larger the flap, the more likely are we to be able to retain a sufficiently large portion of the flap transparent. Another factor is the following: The larger and more even the flap is, the more likely are we to avoid the interference to vision caused by irregular astigmatism. This astigmatism cannot be entirely avoided. I believe that the poor vision in the previous successful cases was due to this irregular astigmatism.

Against these advantages might be brought the disadvantage that the larger the flap the more likely are we to get a perforation in its removal. In my cases, however, although I always removed a deep corneal flap, I had a small perforation in only three cases, due to the marked convexity of the guinea-pig's cornea.

There remains yet the possibility that if the transplanted flap does not heal in, the extensive thinned surface may predispose to the formation of a staphyloma. I have no experience in the human eye, as in the case operated on by me the flap healed perfectly, but in guinea-pigs, where, through undermining by the nictitating membrane, or as the result of infection, the centre of the flap was destroyed, I never saw a protrusion of the remaining thinned portion of the cornea. This might be explained by the marked faculty of guinea-pigs' corneæ to regenerate, a tendency which is not so marked in the human eye.

On the other hand, if the healing is smooth, such a flap, fastened at the four corners, would tend to counteract a beginning staphyloma. In this way, it might possibly be used in keratoconus.

OPERATIONS ON GUINEA-PIGS.

In order to convince myself of the feasibility of the technique of my operation, and to be able to observe the clinical course after the operation, I performed a series of transplantations upon the guinea-pig. It is well known that the guinea-pig is not well adapted to operative experiments on the eye. Wagenmann had, in his autoplastic experiments, in 9 cases, 6 failures through infection; Salzer had 12 serious and 3 mild infections, out of 18 cases. Consequently, I was not surprised to have 5 severe infections out of 12 operations. Added to these, there were 3 cases in which healing was delayed for the first few days in the centre of the flap by the interposition of the nictitating membrane.

At all events, the experiments on the guinea-pigs' eyes demonstrated to me the feasibility of the operation in almost all the cases, and allowed me to watch the clinical course in 4 cases, of which 2 healed with almost complete transparency of the flap, while in the other 2 cases slight infections led to more marked vascularization and only partial recovery of transparency of the flap.

The following is the history of one of the former cases:

Guinea-pig, No. 170. On June 9, 1909, there is mutual transplantation of vertical flap between guinea-pigs Nos. 163 and 170. The operation was performed in the above described manner, without incident. The transplanted flap was 6*mm* wide. At the end of the operation, the flap was absolutely clear. The eye was left uncovered.

June 10. The flap is unchanged, and is everywhere united with the underlying tissue. Its nasal margin has not been undermined by the nictitating membrane. No sign of infection. By daylight the flap appears perfectly clear, but by oblique illumination it is seen that the central third is slightly cloudy, but the iris is seen through, just as through a normal cornea. At one spot there lie, between the flap and underlying tissue, a few coagula. The flap is not shrunken. Its parallel borders appear as delicate crooked lines without opacity. The flap does not lie above the level of the cornea. No vascularization. Scarcely any secretion.

June 12. The flap is unchanged. No secretion. No sign of necrosis or infection.

June 14. Examination with the Zeiss lens shows only a faint nubecula in the central third. Besides this there are

seen minute depressions, over which there is no epithelial defect, however, and which show no opacity. The edges of the flap, which would be most likely to become necrotic, have neither shrunk nor become infiltrated.

June 16. Conjunctival sac clean. Sutures removed.

June 19. The flap is unchanged and perfectly clear, except for a few small grayish-white spots in the lower posterior corner. The margins of the flap can scarcely be distinguished. The epithelium is smooth. A few small vessels enter the flap from above and below.

June 24. The delicate vessels have become more numerous and have advanced toward the centre, accompanied by a delicate opacity.

June 30. The opacity has not progressed. The vessels have reached the edge of the pupil and are retrogressing. The central portion of the flap still shows a few irregularities on its surface, but it does not stain with fluorescein. Even through the peripheral portion the iris can be plainly distinguished.

July 17. Only a few vessels remain in the flap, which is clear and smooth, except for a few grayish-white spots (possibly thickenings).

Aug. 10. The flap cleared up farther. Globe completely free from irritation. At the end of September, the guinea-pig was killed, on account of a large abscess in the snout, in order to prevent the possibility of infection of the eye. The condition of the operated eye was unchanged.

I add to this successful case, the history of the guinea-pig from which the flap was taken for the above case. Healing took place in this case, but on the 10th day, a mild infection destroyed a portion of the centre of the flap.

Guinea-pig No. 163. Received, on June 9, 1909, a corneal flap from animal No. 170. The flap lay in normal salt solution for 30 minutes, before implantation, and did not completely cover the defect. The fixation was successful.

June 10. The flap is in perfect apposition, and does not lie above the level of the surrounding cornea, therefore it is not swollen. The edges can scarcely be recognized. It is slightly grayish, especially in its middle fourth, but does not seem to be infiltrated with pus. The conjunctival flaps are adherent; there is scarcely any secretion in the conjunctival sac.

June 12. The flap is somewhat more opaque, but is adherent to the underlying tissue. It has not been lifted up by the nictitating membrane.

June 14. Condition is the same. Very little secretion. Sutures removed.

June 16. The upper part of the flap becomes somewhat clearer.

June 19. The flap is well attached, but shows a central purulent infiltration, which extends to the floor of the cornea. At the same time, small vessels come in from above and below.

June 25. The peripheral third of the flap does not seem to be involved in the infiltration, but is vascularized.

July 16. The opacity of the flap has receded considerably as well as the vessels.

Aug. 10. The opacity has cleared up more. There are no more vessels to be seen. The pupil and iris can be seen very clearly.

Aug. 17. *Status idem.* Enucleation.

This experiment shows that even with a late infection the flap need not be lost entirely, and that the centre of the flap offers less resistance to infection than the peripheral part, on account of its poorer nourishment. It is not unlikely that the flap would have had better resisting power if it had not lain for half an hour in solution before its implantation.

OPERATION ON THE HUMAN EYE.

For the permission to perform the operation on a suitable case in the human eye, I wish to thank my distinguished teacher Prof. Roemer. The opportunity occurred in July, 1909, when Frederick P. came to the clinic on account of diminution of vision in both eyes. In 1902 and 1903, he was treated here for extensive leukoma of the right eye and traumatic hypopyon keratitis of the left eye. On entrance, there were seen on both corneæ, more marked in the right than the left, numerous deep and faint opacities, going to a variable depth in the cornea. There was an irregular astigmatism. The vision in the right eye was $\frac{6}{60}$ with -1.0 sph. An optical iridectomy promised no result, as the opacities involved the periphery also, and a stenopæic slit with mydriasis caused no improvement in the vision. The fundus was normal. The conjunctiva was clean.

July 21, 1909. Under cocaine anæsthesia, after washing out the conjunctival sac with physiological salt solution, a

6*mm* wide corneal flap was removed along with a small conjunctival flap on each end, without a perforation. Although the knife passed very near the posterior surface of the cornea, two small opaque patches are seen to remain in the posterior layers, beneath the centre. I did not remove more tissue at this point, on account of the danger of a perforation. I allowed the flap to lie in situ, with the lids closed, while I removed from an eye, which was about to be removed on account of sarcoma of the choroid, a corneal flap of like size. This removal occurred without accident, except for a minimal-sized perforation just above the centre, which was the result of my haste to cover the defect in the other eye. The fact that this portion of the flap was thicker than the rest did not seem to influence the result of the operation. The operation on the second eye lasted scarcely ten minutes. The flap was applied to the defect, which it covered smoothly, and, after washing off with normal salt solution, fastened in place by means of four conjunctival sutures. There was no appreciable pain. A wet bandage was applied, and the patient put to bed.

The patient was seen in the evening, when it was seen that the flap lies smoothly in place, and is clear, except for the two small remaining patches. About 1*mm* central to and parallel to the lower limbus is seen the grayish-white scleral margin of the transplanted flap. This is due to the fact that the cornea of the eye of the patient is somewhat larger than the cornea of the enucleated eye. At the margins of the flap are to be seen a slight swelling and opacity of the edge. The rest of the flap does not rise above the level of the remainder of the cornea. The conjunctival flaps lie in good apposition and are slightly swollen. No purulent secretion.

July 22. The flap remains clear, even the edges not being affected. The conjunctival sutures are clean. A little mucous secretion. No pain. Patient promptly counts fingers.

In the next few days, the condition remained unchanged.

July 26. As there was always photophobia when the bandage was removed the patient was given a nickel plate with a central opening.

Aug. 5. Marked conjunctival injection about the conjunctival flaps, which have completely united. From this portion of the limbus, numerous small vessels pass into the posterior layers of the flap, about 2*mm*. Here the posterior lamellæ are slightly opaque, and show in the posterior inferior angle 2 or 3 small grayish-white points, which, however, do not seem to be actual infiltrations. The rest of the flap is clear. There is no groove to be seen at the margins,

and the flap does not seem to have shrunk. The flap does not rise above the level of the rest of the cornea. The epithelium is everywhere present, and the surface smooth and shiny. During the examination, there is some lachrymation, but there is neither ciliary injection nor iridic irritation. The iris and interior of the eye can be seen perfectly clearly through the flap.

Aug. 10. The vessels at the upper and lower limbus have extended somewhat farther centripetally, along with the delicate opacity of the deeper layers of the flap. Even with the help of the Zeiss lens, one cannot be sure whether these vessels lie on the under surface of the flap or on the posterior surface of the cornea. The pupil and iris can be clearly seen. The three grayish-white spots in the inner-lower corner can scarcely be distinguished with the lens; a little grayish stringy secretion consisting of leucocytes. Patient does not complain. A Fuchs wire screen is applied. Irrigation three times daily with boric acid solution. Over night, white precipitate ointment.

The flap is still anæsthetic; the remaining portion of the cornea, hypersensitive.

Aug. 25. In the last few days, the patient complained a good deal of lachrymation and itching of the right eye, as well as dazzling. On the use of boric acid the symptoms rapidly disappeared. The eye is now white and not irritable. There is no secretion. The patient holds the eye well open.

Sept. 1. The few deep vessels which extend from the upper and lower limbus have extended farther toward the centre, but they leave the middle quarter of the flap free.

Sept. 25. Patient discharged as ambulant case. Given boric acid.

Dec. 19. The flap is completely healed in The conjunctival flaps can scarcely be distinguished. The globe is white and not irritated. The patient had at no time any pain. The flap has not become smaller, and lies level with the rest of the cornea. The border lines can be distinguished only in places with the lens. With ordinary illumination, no evidence of operation can be seen on the eye; 1*mm* centrally from the lower limbus, can be seen the narrow margin of the transplanted sclera. There are no vessels to be seen, except in the upper third; a few long vessels can be distinguished with the lens. The flap is no longer anæsthetic. Vision, right, is $\frac{3}{15}$, with -1.00 D. spher.

April 16, 1910. The right eye is completely white and not irritated, and can be held wide open even in bright light. The corneal flap is unchanged. Its sensibility is somewhat less than that of the rest of the cornea.

Now it is a year since the operation, and there have been no further changes in the eye. I believe that this result may be considered final.

The following laws may be laid down for homoplastic operations:

1. Other things being equal, a transplantation has the best prospects of success when the flap is carried over into tissue of the same kind, *i. e.*, when the conditions for its further existence are physiological.

2. Between the flap and the underlying tissue there is formed a fibrinous layer into which vessels and polynuclear leucocytes force their way from the underlying tissue.

3. In the transplanted tissue, the greater portion of the cellular elements, and possibly also the intercellular substance, are destroyed. From the cells which survive there is set up a regeneration, which is kept up by the formation of blood-vessels and cell-proliferation from the surrounding tissue.

4. It is often difficult to decide how much of the regenerated tissue is formed by proliferation of the surviving portions of the transplanted tissue, and how much by wandering in of cells from the neighborhood.

A microscopic examination of the eye of guinea-pig No. 170, ¼ year after the operation, shows some interesting facts.

A horizontal section of the cornea shows, with a lens, almost a normal cornea. The curvature is normal, and the surface smooth. In the central portion, where the flap was implanted, it is very little thicker than in the peripheral portions, which corresponds to the normal condition.

Under the low power of the microscope, it is seen that the cornea is covered by a stratified layer of epithelium, which is of varying thickness, with irregularities on the surface as well as protrusions of epithelium into the substance of the cornea. The epithelial layers are not as uniform as usual. The most marked thickening of the epithelium (about 7–9 layers) occurred at the margins of the transplanted flap. From the position of this epithelial thickening, it is seen that the flap has not shrunk. It takes up ¾ of the width of the cornea.

The flap is firmly united to the underlying tissue everywhere, from which it cannot be distinguished. The only thing which distinguishes the transplanted tissue is the fact that it contains more nuclei and takes up more eosin in the hæmatoxylin-eosin stain. With the Van Gieson and carmine stains there is no such difference.

With the high power, it is seen that the endothelium, Descemet's membrane, and the posterior layers of the parenchyma have remained intact. In front of this is a zone, in which are seen the cross-sections of very small blood-vessels. These correspond to the layer of union. These vessels send no branches, either forward into the flap, or backward into the underlying tissue. A few of these vessels are accompanied by scattered connective-tissue cells. In the layer of union, there is no evidence of necrosis, fibrin, or leucocytes. In the flap, the lamellæ are less regularly arranged, and appear, in places, more wavy and thicker.

The histologic findings give the impression that in this method the degenerative changes in the transplanted flap are very slight.

CONCLUSIONS

1. The removal of the flap in the above described manner is accompanied by very few degenerative changes.

2. The fixation of the flap is sure, and an injurious effect from winking is excluded.

3. The nutrition of the flap is favored by the large wound surface, and especially by the transplantation of a large portion of the limbus, thus allowing a rapid restoration of the normal nutritive changes.

4. In agreement with the slightness of the degenerative changes, there are only slight vascularization, infiltration, and formation of new connective tissue.

5. The extent of the flap allows us to hope for a good optical effect, even when all of the opaque tissue has not been removed, or when portions of the flap become opaque.

6. Even with perfect healing of a partial flap of the cornea, we can never expect to get perfect vision, because the irregularity of the epithelium and parenchyma, even without opacities, produces an effect like an irregular astigmatism.

7. Where we want it for its protective effect, the method is of value, on account of the good fixation of the flap and its large surface.

EXPLANATION OF THE PLATES

Plate 1. Horizontal section through a guinea-pig's cornea, with transplanted flap. (Guinea-pig No. 170.) Appearance with lens. The epithelium is somewhat uneven and irregular. The two points of greatest thickening mark the lateral boundaries of the flap. The lower boundary of the flap is indicated by 6–7 cross-sections of vessels. The greater wealth of nuclei in the transplanted tissue can be seen.

Plate 2. From the same cross-section, near the centre of the cornea. Hæmatoxylin-eosin. One can distinguish the irregular layers of epithelium, and the wavy arrangement of the lamellæ in the transplanted tissue. It has taken the eosin stain stronger than the remaining deeper layers of the cornea, from which it is separated by the lumen of a vessel. The transplanted tissue shows more fixed corneal cells and few wandering cells.

LITERATURE.

In regard to the literature, one can consult the references in F. MARCHAND, *Prozess der Wundheilung*, 1901, and SALZER, *Zeitschrift für Augenheilk.*, 1900, vol. iv., p. 142.

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REPORT OF THE PROCEEDINGS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

By C. DEVEREUX MARSHALL, F.R.C.S.

An ordinary meeting at the Medical Society's Rooms was held on Thursday, 25th of January, under the presidency of Mr. J. B. LAWFORD. It was announced that the Society had accepted the invitation of its Dublin members to hold the May meeting on the 11th of that month in Dublin.

Mr. A. C. ROPER showed a case of **melanotic sarcoma of the conjunctiva** with secondary nodules in the skin. There was no trace of anything in the fundus. He was advised to radically remove the whole of the orbital contents. The skin nodules were found not to be malignant. Mr. G. COATS showed (1) **concretions (Drusen) of the papilla;** (2) **corpora amylacea in the retina.** Dr. FARQUHAR BUZZARD said the appearance was different from corpora amylacea in nerve tissue generally, as in this case there was a definite capsule. Mr. RAYNER BATTEN showed a **fixation fork,** designed for the insertion of sutures in the sclerotic in advancement operations. By its aid one could pick up exactly the amount required, and there was no danger of the needle being pushed in too deeply. He also showed a case of **central (macular) coloboma of the chorioid** of unusual form, symmetrical in the two eyes. Mr. L. PISANI showed a case, which was originally exhibited in 1880, with **peculiar bodies on the iris after needling** for lamellar cataract. Mr. MAYOU thought the bodies might be due to implantation of the cells of the capsule on the iris. Mr. GREEVES showed (1) a case of **metastatic septic chorioidoretinitis,** and (2) a case of **quinine amblyopia.** Mr. COATS showed a case of **tubercle of the chorioid** in a cat. Mr. MAYOU

related a case in a cat also, which bore out Mr. Coats's remark. In that case, however, there was a small nodule in the breast. He thought it was not likely for primary tubercle to start in a chorioid without there being a lesion elsewhere in the body. Mr. WRAY showed a case of **glaucoma,** a case of **keratitis disciformis,** and one of **exostosis of the roof of the orbit.** Mr. R. R. JAMES showed a case of **hyaline degeneration of the disk,** and Mr. HORSFORD a case of **mal-development of the eyes in a child.**

Mr. A. A BRADBURNE read a paper entitled **Hereditary ophthalmoplegia in five generations.** In this family there was a condition of ptosis accompanied by an almost complete loss of the ocular movements, which had been present in five generations. After enumerating the numbers affected and briefly describing the condition in each, he proceeded to consider the various types of this rare affection. He classed anomalies of congenital ocular immobility into three divisions: first and most common, those in which only ptosis was present. Secondly, when this was combined with epicanthus. Thirdly, a type in which ptosis might or might not be present, but in which the ocular movements were very defective or altogether wanting. This last type embraced three sub-divisions: (*a*) ptosis associated with defect of the superior rectus; (*b*) more extensive involvement of the eye muscles with or without ptosis, the condition being due to some nerve lesion; (*c*) this type included the same kind of matters, but the condition was due to entire absence or very defective development of the muscles. His own case came under this category. The author pointed out that in two of the younger members a certain return of the movements had occurred, and suggested how this might have come about. He showed that in the shark the ocular muscles arose from three centres: one for the superior obliques, one for the abductors, and the remaining four muscles arose in pairs from a third. These paired muscles were the superior with the internal rectus and the inferior rectus with the inferior oblique. In all the members of this family the superior obliques were probably present, and hence if any further movements became possible one would expect their appearance to follow somewhat the lines as seen in the shark. In one of the members of the family it had done so,

as one eye was able to move from a point straight in front outwards, thus seemingly pointing to a development of the second centre. But that did not follow in another case in which both eyes possessed full lateral motion. By analogy one would expect to find development of motion in the upward direction, but such was not present. Hence the human development was from a different centre to that of the shark, or another factor was present in the case. As the patient had ptosis, motion in an upward direction would not be of much practical use, hence it seemed as if the visual act had had something to do with the "evolution." This seemed probable as, in the same case, to prevent diplopia, the visual act had produced a convergent squint. Finally, he asked if the argument could be logically reversed and the condition of muscle palsy attributed to a long-continued non-use of the eyes in some ancestor afflicted with ptosis. The paper was discussed by Mr. BISHOP HARMAN, who pointed out that in the sunfish each of the recti, except the rectus externus, sent off a separate slip which went to the circular fold round the eye, and was capable of retracting a fold from the cornea, and one saw the original development of the levator. The idea of an arrest of development running through a family was easier of acceptance than the more difficult theory which the author had brought forward. Mr. BEAUMONT (Bath) suggested the condition might not be so much a pathological one as a tendency in this family to a throw-back to a primeval condition antecedent to the development of the oculo-motor muscles.

Mr. GEORGE COATS read a paper entitled **On crystal-like bodies of radiate structure in the lens.** He said they were found in a hypermature cataract in a woman aged 69, and had not been seen clinically. In the lens nucleus, which had undergone but little degeneration, round and oval bodies, measuring from 0.035 to 0.160*mm* were found. They had a refractile crystalloid appearance, and were marked by numerous fine lines converging towards an axial linear streak or spot. There was frequently a tendency to cleavage along these lines, so that the body was split up into a number of radially disposed pyramidal sectors, not unlike the pyramids of the kidney. In the larger crystals there was a central tuberculated mass of mineral hardness, the structure resembling that of a rubber-

cored golf ball. Usually the adjacent lens fibres were arranged round the crystals to form a kind of capsule. Only two similar observations were on record, in one of which the bodies were seen clinically as globular beads with a silky or pearly sheen. In all these cases the lens was cataractous; in two the opacity was senile, in one lamellar, the patient being a child of six. In all, the crystals were confined to the more central layers of the lens. As to the nature of the crystals, the scarcity of material made it impossible to carry out extensive histochemical researches, but they showed considerable structural resemblance to leucin, a diagnosis which was sufficiently probable, since the crystals occurred in cataractous lenses, and leucin is a product of proteid disintegration. They occurred, however, in the least degenerate part of the lens. Hydrochloric acid produced no effervescence. Dr. W. H. WILLCOX had kindly examined the specimens, and reported that the appearance of the bodies was "similar to that which leucin takes when it has been subjected to dehydrating processes."

The PRESIDENT asked whether Mr. Coats found these bodies in the extracted lens, *i. e.*, whether the eye had been removed. Mr. BURDON-COOPER (Bath) asked if Mr. Coats had ever found tyrosin in the lens. He had himself discovered that substance in the aqueous after needling for lamellar cataract. Professor HALLIBURTON, of King's College, confirmed that it was tyrosin. In wondering how tyrosin could have got there it occurred to him that possibly it was the result of hydrolysis of the lens, and he therefore sent for a pig's eye at the butcher's, and found the lens yielded almost pure tyrosin. The microscope gave the most delicate test for it. Tyrosin was fairly soluble in sulphuric acid. He had found the substance in the lens in all cases of cataract, and he was strongly of opinion that the change which took place in senile cataract was a hydrolysis of the lens. Mr. COATS, in reply, said there was nothing in his sections which resembled tyrosin.

A CLINICAL EVENING WAS HELD AT THE MEDICAL SOCIETY'S ROOMS, ON THURSDAY, FEBRUARY 8, 1912, THE PRESIDENT, MR. J. B. LAWFORD, IN THE CHAIR.

Mr. ROBERT DOYNE showed a case of **guttate iritis.** He said he had never seen a case in a man. The guttæ were

independent, in position, of the inflammatory points. The guttæ, within a few days, might have completely changed their position. In this patient a week ago there were four well-marked spots, but now there was only one. It seemed to be an exudation between the layers of epithelium where it was reflected; usually the subjects of the condition were gouty. The PRESIDENT reminded members of the cases shown by Mr. Doyne two years ago, and they seemed to have occurred only in cases of chronic iritis. If the condition happened in acute iritis it might be missed in the general haze.

Mr. CHARLES WRAY showed three cases of **frontal sinus disease,** with pathological specimens; also a severe case of **ingrowing lashes treated by the electro-cautery.** He said that although Ziebler advocated that for entropion the cautery should be at a white heat, his own experience was that it was better at a dull-red heat; and he preferred the thermo-cautery.

Mr. WRAY also showed a **cyclo-phorometer** and described his method of testing. The case was discussed by Mr. WORTH, who referred to a book he published ten years ago on the subject.

Mr. G. COATS exhibited a case of **unilateral proliferation of the uveal pigment,** and Mr. TREACHER COLLINS one of **unilateral melanosis of the uvea and sclera, with elevations on the affected eye.** These were discussed together. Mr. COATS said there was evidence that the spot was there shortly after birth, and it had increased considerably in the last year. There was increased pigmentation of iris, fundus, and sclera. The point of most interest was the presence of elevations on the surface of the iris; 26 cases of the kind had been recorded, and of those no fewer than 7 developed sarcomata in the eye late in life. The commonest age for it to occur was between 50 and 60, and the age of the youngest case recorded was 34.

Mr. COLLINS said he had his case under observation and care, backwards and forwards, for ten years. Not every case of melanosis showed elevations on the iris. Some years ago he showed, at the Society, sections at the seat of melanosis, apparently dating from birth, in a man æt. 60. He thought it was melanotic sarcoma of the ciliary body. He recently re-examined that section and found it remarkably free from elevations.

Mr. HEWKLEY raised the question whether it was the rule for the irides to be a light-gray color at birth.

Mr. LESLIE PATON showed a case of **hole in the macula,** which he considered differed from ordinary cases of hole in the macula, and from the 15 cases collected in Mr. Ogilvie's paper, in the fact that the hole was bluish white. There was no history of injury in the case, yet there was some evidence that it was a recent occurrence. There had evidently been considerable retinal disturbance all round the macular region. Apart from the hole, the general appearance was like that of albuminuric retinitis, but there was no trace of albumen in the urine, and no other organic disease.

Mr. GREEVES showed again a patient with **recurrent unpigmented tumor of the sclera,** the case having been brought forward originally in October last. Removal was done, and later there seemed to be a recurrence, but the scar tissue which began to form seemed to strangle the tumor, and it had now disappeared. The first tumor was pronounced to be a very cellular hæmangioma.

Mr. CUNNINGHAM showed two cases: (1) **cholesterine crystals in the anterior chamber;** (2) **retinal hemorrhage and exudation in a young subject.** The second case he regarded as one of vascular disease.

The PRESIDENT, discussing Mr. Cunningham's second case, said he saw the lad in hospital, and no condition had been found which was likely to be causally related to the eye condition: nothing, for instance, in the blood-cell count, or the coagulation time, or the presence of any bacterial condition. The possibility of lead poisoning had also been excluded.

Mr. DOYNE considered that the case belonged to the class which Mr. Eales had worked at, and which he called spontaneous hemorrhages, but which had not had much light thrown on them. He tried to find out something about the condition years ago, and was convinced that the veins, not the arteries, were responsible for the hemorrhage. The cases seemed to be limited to men, but not to young men, as Eales thought. The whole eyeball might appear to be suddenly filled with blood. The cases which he had seen, recovered, sometimes with very good vision, leaving some silvery stains

in the retina, and sometimes some membrane in the vitreous. In one case he found that the blood tension was very low. Von Pirquet was positive.

Mr. HARRISON BUTLER said Professor Axenfeldt had proved several of these diseases to be due to alterations in the retinal vessels, and he pointed out that nearly all the cases had a tubercular basis.

Mr. LESLIE PATON did not agree that these cases were analogous to cases of intervitreous hemorrhage. One case he saw had some cranial nerve palsy.

Dr. FARQUHAR BUZZARD said he knew the case to which Mr. Paton alluded. It was not a case of true Bell's palsy, because only certain fibres of the facial nerve were affected. He thought it might have been due to a local hemorrhage in the facial nucleus or the facial nerve. One could easily imagine, following on Mr. Doyne's suggestion, that it might be caused by a small tubercular deposit in the pons.

Mr. CYRIL WALKER referred to the case of a man, æt. 21, who was healthy, but the sight of one eye was bad. He had many streaky-looking hemorrhages in the nerve-fibre layer, and there were ten fairly large ones, the largest a quarter the size of the disk, and there were twenty smaller ones. In three weeks the sight improved a great deal. There was no macular star. Two months later the patient had a very large intra-ocular hemorrhage, which blotted out everything. That remained opaque until two months later, when it began to get translucent. The treatment was mainly rest, and the patient was also given some mercury.

Mr. LAYTON DAVIES related the case of a male clerk, æt. 19, who came complaining of severe headaches, and that a few days previously he was blind in his left eye. In the fundus, striate hemorrhages were found over the retina, and dilatation of the veins, with white exudation in the macular region. It looked like a case of albuminuric retinitis, but a physician found the urine normal.

Mr. W. H. JESSOP considered such cases were somewhat allied to Raynaud's disease, or even hæmophilia: the subjects of it, when struck, rapidly became bruised. There seemed to be some defect in the coat of the arteries, leading readily to transudation.

The discussion was continued by Mr. RAYNER BATTEN and the PRESIDENT.

Mr. DAWNAY showed a case of **persistence of anterior portion of hyaloid artery.**

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. MARTIN COHEN, Secretary.

MEETING HELD MONDAY, DECEMBER 18, 1911. DR. H. W. WOOTTON IN THE CHAIR.

Dr. Edgar S. Thompson presented a patient with **tuberculosis of the iris.** Male, 21, single. Previous history negative. Complains of indefinite pains in the left eye for two weeks. Health apparently good. Physical examination negative. Von Pirquet and Wassermann negative. Mixed treatment had no effect on the lesion. Vision of left eye $\frac{20}{50}$, improved with glasses. Vision of right eye normal. Oblique illumination of left eye showed the cornea to be normal. Pupil irregular, due to posterior synechiæ. At the inner and upper quadrant at the root of the iris there is a small solitary grayish tubercle, protruding into the anterior chamber; with the aid of a corneal loupe, blood-vessels could be demonstrated in the tubercle.

Dr. Cutler, in discussing the case, reported the case of a woman who had trachoma with dacryocystitis, perforating ulcer of cornea, and three minute grayish nodules emanating from the root of the iris. Physical examination of the lungs and sputum proved tuberculosis; the Von Pirquet was positive.

Dr. Koller said that he agreed with Dr. Thompson as to the diagnosis of tubercle of the iris. This affection frequently appeared more under the picture of an exudative iritis which after healing left numerous adhesions and relapsed after irregular intervals. It seems by no means as rare as was formerly believed. In this locality, the typical form where the

iris appears to be studded with tubercles, seems to be rare. Dr. Koller has never observed any cases here, but in a German clinic he saw two cases within one week. The diagnosis, except in typical cases, is by no means easy. The general condition of the patient is mostly surprisingly good and does not give any clue as to the nature of the trouble. (According to Krueckmann, tuberculosis of the iris does not occur in consumptives or in cases of tuberculosis of the joints, but mostly in conjunction with tuberculous glands, chiefly of the mediastinum and the mesentery.) Diagnostic tuberculin injection would not go far to clear the matter, as a local reaction can rarely be elicited except by large doses, and is besides not desirable; and a general reaction is not decisive considering the widespread occurrence of tuberculosis. If we have a case of plastic iritis, perhaps with many old adhesions, and if a negative Wassermann reaction makes the diagnosis of syphilis unlikely, we should at least think of tuberculosis as the possible etiology. The suspicion is strengthened by evidences of fresh vascularization, such new blood-vessels tending towards and ending in not well defined foci within the stroma of the iris. A particularly characteristic sign for tuberculosis is the location of nodules at the root of the iris, protruding into the anterior chamber from its angle and resulting in the formation of a localized mass of gray connective tissue.

Dr. THOMPSON, in closing the discussion, said that connective tissue formation occurs later in the disease, and expressed the view that this patient might recover without any treatment.

Dr. JAMES W. CASSEL reported a case of **Parinaud's conjunctivitis.** Josephine Z., 25, appeared at the Manhattan Eye, Ear, and Throat Hospital, November 27, 1911, saying her right eye had been "sore" for about 10 days. No history of fever, accompanying the attack, could be elicited. Preauricular glands were moderately swollen. Submaxillary glands and the entire submaxillary region were greatly swollen. Conjunctiva inflamed and swollen. Soft and velvety granulations with a grayish deposit in the centre were present on the conjunctivæ of both upper and lower lids and in the retrotarsal folds, but not on the eyeball. A culture proved negative; so did the Von Pirquet and the Wassermann tests.

Under local cleanliness and argyrol, with potassium iodid

internally, the granulations became smaller and the conjunctiva less swollen.

No history was obtainable of association with animals. Patient is well advanced in pregnancy and, with the exception of no definite history of fever and constitutional disturbance at the onset, she presents the typical appearance of Parinaud's conjunctivitis.

Dr. WEIDLER presented 3 patients. The first was a case of **homonymous hemianopsia.** A. L., 60, white, barber, came to the Manhattan Eye and Ear Hospital on November 15, 1911, complaining of partial loss of vision in both eyes for past two weeks. Except for a chancre 30 years ago, which was treated, the general health was good. Two weeks ago he fell from a street car, but does not think he struck his head. Got up himself and walked away unassisted. Two days later he could see only the half of things. Station is uncertain, gait good, knee-jerks slightly active, co-ordination fair, grip good and strong. Urine and Wassermann negative, blood pressure 160. Reaction to light and accommodation. No change in the fundus. The fields show a complete **homonymous hemianopsia,** not involving the macular fibres.

One month later the field for the left eye was greatly improved and enlarged, but remained unchanged in the right.

The second case was one of **microphthalmus.**

Wm. S., 6, birth normal. Mother died of tuberculosis at 29. No eye trouble in any member of the family. Light-colored, sparse hair appeared first two years ago. No noteworthy defects of physical or mental development. Head measures 20½ in. in circumference, 10¾ in. over top from ear to ear. Eyeballs small. Corneæ 7*mm* in horizontal and 8 in the vertical axis. Palpebral opening 21*mm* across. Lateral strabismus. Pupils 2.5*mm*, irides blue and react to light, accommodation, and convergence. Disks are a trifle gray, and the chorioid shows an under-development. There are a number of grayish threads of pupillary membrane which extend to pupillary edge.

The third case referred to **medullated nerve fibres at some distance from nervehead.**

B. S., 26, came to the clinic because of poor vision. Down and out from macula about 3 p.d. from nervehead is a fan-

shaped area of opaque nerve fibres, which cover the retinal vessels slightly.

Dr. WEIDLER again presented the case of **lupus erythematosus,** which was shown at the previous meeting, so that the treatment with carbonic acid snow might be followed.

In the discussion:

Dr. WOOTTON asked whether Wernicke's hemiopic pupillary reaction was present.

Dr. LESZYNSKY mentioned a case where there was a reversal of Wernicke's phenomenon.

Dr. THOMPSON had never seen opaque nerve fibres at such a distance from the macula.

Dr. HELLER reported a case where one eyeball was very small and the other large.

Dr. LESZYNSKY thought the case of microphthalmos, if more minutely examined, would show other stigmata of malformation.

Dr. WOOTTON mentioned a case with small eye and cornea, an arcus senilis, and head malformation.

MEETING HELD MONDAY, JANUARY 15, 1912. DR. JOHN R. SHANNON IN THE CHAIR.

Dr. WOOTTON presented a case of **chronic nuclear palsy.** The patient, a man 28 years of age, first suffered from diplopia about two years previously. At the present time he presented incomplete bilateral ptosis, incomplete paresis of the third, fourth, and sixth cranial nerves. Rotation inward, upward, downward, and outward was markedly limited in both eyes. There was no pupillary disturbance, and the accommodation was unaffected. Convergence was also preserved. The fundus was normal, the fields of vision normal. The patient denied syphilis and the Wassermann reaction was negative. The vision was good. There had been no cerebral symptoms at any period during the progress of the disease.

Dr. WOOTTON also presented a case of **chorioiditis disseminata** commencing at the eighth year and resulting in blindness at the thirteenth.

The patient was a boy 13 years of age, and an only child. The mother's eyes were normal. An examination of the father could not be obtained. Wassermann reaction was

strongly positive, and it was stated by the parents that the disease had been checked temporarily on previous occasions by inunctions. The fundus presented extensive and numerous patches of chorioidal atrophy. Both retinæ were atrophic and both disks showed secondary atrophy. Vision in the right eye was nil, in the left eye was eccentric and equalled fingers at from 4 to 8 feet.

Dr. LESZYNSKY thought that Dr. Wootton's patient of nuclear palsy was a case of double ophthalmoplegia externa coming on acutely and became progressive. This condition is sometimes due to alcohol or toxæmia. Tabes is not necessary in these cases.

Dr. H. H. TYSON reported a case of methyl alcohol amblyopia caused by inhalation.

A young man, G. K., æt. 21 years, was employed on December 3 and 4, 1911, shellacking the interior of a large beer vat. On December 5 he complained of nausea, headache, and vertigo, unsteady gait, and was unable to work.

December 7. Vision commenced to fail and he consulted his family physician who made a diagnosis of methyl alcohol poisoning and prescribed laxatives. Vision continued to fail and he was advised to consult an oculist.

He visited the clinic of the N. Y. Ophthalmological and Aural Institute on December 12.

Examination: Vision eccentric R $\frac{1}{200}$, L $\frac{2}{200}$ (counting fingers in extreme inferior temp. fields). Pupils widely dilated with practically no reaction to light or convergence. Eyes painful (behind globes) on rotation. Fundi showed optic neuritis with dilated dark veins, arteries darker than normal, but not altered in size, with extensive retinal œdema especially marked along the vessels.

He was admitted to the hospital and was given pilocarpin sweats, and calomel and jalap as purgatives, with strychnia.

Blood examination by Dr. Shook: Red blood corpuscles 5,265,000, white blood corpuscles 8000. Hemoglobin 106%.

He remained in hospital 8 days. On December 19: Vision R $\frac{1}{200}$, L $\frac{2}{200}$, absolute central scotomata extending 30° about fixation point in all directions, no color field. White field contracted about 10°. Strychnia continued.

Two weeks later, January 3, 1912, vision showed remarkable

improvement to 20–30 each size. No central scotoma could be discovered by perimetric or Bjerrum test. White fields about normal. Color fields:

R. Blue 20 Sup. 30 T. 30 Inf. 10 Nasal.
R. Red and green 8 Sup. 8 T. 2 Inf. 5 N.
L. Practically concentric contraction.
Blue 40 Red and green 15 Sup. and Inf. and 25 Temp. and Nasal.
Reads Jaeger No. 1 at 10″ each eye. (L clearing.)

Retinal arteries contracted, veins dark, pallor of disks, edges still swollen and blurred. As to how much vision will be maintained permanently, only the future will determine. As a rule a recession of about one-half should occur.

In this connection it is of interest to note that of two of his fellow co-laborers who were employed on the same work, but who had labored three days each during same time, one died suddenly 48 hours and the other 72 hours after stopping work; also that at the same brewery last year, with the same lot of shellac, five laborers had died with symptoms of methyl alcohol poisoning.

Dr. WOOTTON, in discussing Dr. Tyson's report, said there were no cases on record of amblyopia due to inhalation of wood alcohol in Great Britain owing to the fact that it was not allowed to deodorize wood alcohol. He also claimed that there is imperfect ventilation for the workman employed in shellacking beer vats.

Dr. LESZYNSKY asked if denatured alcohol, which is sometimes used in bathing children, has caused defects in vision.

Dr. N. L. WILSON asked if there was any law in regard to the manufacturing of wood alcohol.

Dr. WOOTTON said that there was no law prohibiting the substitution of Columbian spirits in hair tonics or bay rum.

Dr. TYSON closed the discussion by stating that there was no fresh air supply in the beer vats for his patient.

Dr. O. SCHIRMER presented a case of **leprosy of the eye,** which he had presented a year ago, when there was a leprous nodule in the left sclera with adjoining interstitial keratitis. When operated upon soon afterwards, the nodule could not be radically removed, since a root was found to pass through the sclera into the ciliary body. Nevertheless, there has only

been an insignificant relapse, the new nodule after a year's growth being only half its former size. The diffuse infiltration of the cornea has made no progress. The excised nodule was examined microscopically. It consisted of typical leprous tissue with enormous quantities of bacilli. Furthermore, there has started in the centre of the right cornea a typical leprous keratitis consisting of about a dozen very minute gray infiltrates, which are situated in different layers of the cornea; there is no diffuse haziness around them nor any irritation of the eyeball. Leprous nodules have developed in the iris, two in each eye, situated in the minor circle; their color is brown, and their size that of a pinhead. There are no signs of inflammation or irritation, the surrounding iris tissue being absolutely normal, in contour sharp, no synechiæ having been formed. The treatment during the previous year consisted of nastin injections and X-ray applications. Nevertheless the leprosy has made great progress all over the face. The patient has now the typical facies leonina.

Dr. WOOTTON, in discussing Dr. Schirmer's case, said that no case of leprosy had ever developed in this country.

Dr. WEIDLER asked if there was a microscopical examination made of the leprosy nodule and also if cultures were made in the case of concretions of the lower canaliculus.

DR. SCHIRMER replied that the microscopical examination demonstrated the usual leprous tissue, but cultures were not made of the canalicular concretions.

Dr. O. SCHIRMER reported a case of concretion in the lower canaliculus with microscopical demonstrations. The specimen demonstrated came from the right lower canaliculus of a girl 20 years old who had been suffering from discharge from this eye for two years. The clinical features were: The ordinary slight conjunctivitis, especially of the lower fold in its nasal half, slight inflammatory œdema of skin and conjunctiva over the lower canaliculus, which contained yellowish pus. Upper canaliculus and sac normal. In the slit canaliculus were found two concretions the size of half a pea, yellowish gray in color and soft in consistency. After they were removed, the eye became normal in a few days. No other treatment than lukewarm compresses was given. The specimen shows the ordinary thin, slightly tortuous branches with ramifica-

tions beautifully stained with Gram. Exact diagnosis as to species of the fungus cannot be made, as there are no ramifications, which do not develop except in cultures. As to the name, the author, following the classification of Petruschky in the *Handbuch der pathogenen Mikroorganismen*, uses the name "Trichomyces" (hair fungus), which includes as a higher order the different species in question, namely, leptothrix, streptothrix, actinomyces.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE THIRD AND FOURTH QUARTERS OF THE YEAR 1910.

By Dr. G. ABELSDORFF, in Berlin; Prof. ST. BERNHEIMER, in Innsbruck; Dr. O. BRECHT, Prof. R. GREEFF, Prof. C. HORSTMANN, and Dr. R. SCHWEIGGER, in Berlin; with the assistance of Prof. A ALLING, New Haven; Prof. E. BERGER, Paris; Prof. CIRINCIONE, Genoa; Dr. DALÉN, Stockholm; Prof. HIRSCHMANN, Charcow; Dr. J. JITTA, Amsterdam; Mr. C. DEVEREUX MARSHALL, London; Dr. H. MEYER, Brandenburg; Dr. P. VON MITTELSTÄDT, Metz; Dr. H. SCHULZ, Berlin; Prof. DA GAMA PINTO, Lisbon; and others.

Edited and Translated by Dr. MATTHIAS LANCKTON FOSTER.

(*Continued from page 202.*)

Sections XX.–XXIII. Reviewed by Dr. H. MEYER, Brandenburg.

XX.—RETINA AND FUNCTIONAL DISTURBANCES—*Continued.*

DOR (347, **Treatment of detachment of the retina with tuberculin**) believes tuberculosis to be an etiological factor in many cases of retinal detachment, and has accordingly treated five cases with tuberculin, with a good result in three. The usual treatment with injections of salt was also used. In one unsuccessful case, photopsias appeared morning and evening after the injection of tuberculin, which he ascribed to a reaction produced in the detached retina by the tuberculin.

CAUSÉ.

A glioma may be simulated by a metastatic disease of the chorioid, of the retina, or of the vitreous, tuberculosis of the chorioid, simple detachment of the retina, certain congenital anomalies, like a persistent hyaloid artery, and, very rarely, by a subretinal cysticercus. CURTIL (348, **Metastatic oph-**

thalmia simulating glioma of the retina) saw two cases of pseudo-glioma caused by metastatic irido-chorioiditis. In the first case, a child of 3 years, it was preceded a few months before by an alimentary intoxication, allantiasis; in the second case there was no history of an infectious disease. The metastatic or tuberculous chorioiditis produced a typical pseudo-glioma, a slow disease with a fluid, yellowish exudate, which detaches the retina and is localized behind the lens, whence the characteristic reflex is obtained. It is a metastatic embolism by microbes that are not pyogenic. There are no positive differential diagnostic signs between glioma and pseudo-glioma; both diseases exhibit the same variations as regards the intraocular tension, the inflammatory symptoms, and the appearance. The age of the patient alone can give a certain degree of assurance, as glioma rarely occurs after the seventh year. CAUSÉ.

BERGMEISTER (349, **Contribution to the question of glioma**) describes the microscopical condition found in the gliomatous eye of a child twenty months of age. Three things seemed to indicate the persistence of fœtal conditions, referable to the fifth and seventh months of fœtal life: The abnormality of the pars plicata of the ciliary process, which appeared to be shoved forward on the posterior surface of the iris; the condition of the remains of the retina that were intact on the tumor; and the presence of vitreous vessels of embryonal character.

HARBRIDGE (350, **Sudden obstruction of the retinal circulation**) reviews three cases and describes the phenomenon as due primarily to arteriosclerosis. Case 1 showed frequently recurring spasms of retinal circulation in both eyes, accompanied by complete blindness for about four minutes, and covering a period of eleven days. The contraction of the arteries was observed with the ophthalmoscope. There has been no recurrence. There was a moderate amount of sclerosis, but no specific or rheumatic history. Case 2, obscurations of half the field of vision, recurring at frequent intervals during a period of fourteen months. Followed by recovery. Moderate arteriosclerosis with a heart lesion. Case 3, attacks of hazy vision after exertion, followed by attacks of rapidly recurring obscurations in one eye, which continued to

absolute blindness. There was a cherry-red spot at the macula. A curious cycle of vascular changes could be observed in this case. The vessels were seen to be emptied of blood and then refilled with broken columns. ALLING.

In the case described by BAHN (351, **A rare anomaly of the inferior vein of the retina**), a vein which was abnormally large bifurcated at the lower border of the disk into two short separate branches which penetrated the disk at two points about one-eighth inch apart. ALLING.

Five cases are reported by KNAPP (352, **Intermittent closing of retinal arteries**) as examples of this condition. (1) Age 66. Acute glaucoma in both eyes. Successful iridectomies under ether. Recovery of vision in one eye and blindness in the other, the ophthalmoscope showing atrophic disk with arteries narrow and variable in calibre. (2) Age 53. Permanent sudden blindness in one eye without preliminary obscurations. Temporary obscurations in the other eye. Retinal arteries sclerosed and irregular in calibre. (3) Age 32. Migraine-like attacks for many years, followed by sudden loss of vision in the lower half of one field. The superior retinal artery small and wax-like. Later obscurations in the upper half of the other eye. (4) Age 60. Sudden loss of vision in one eye consequent upon emotional excitement. Return of sight. The inferior temporal artery obliterated at one point. (5) Age 63. Sudden blindness in both eyes. Gradual improvement to $\frac{4}{200}$ and total blindness again. Retinal œdema and hardened arteries. ALLING.

BRUNS'S (353, **A spasm of the retinal arteries**) case was a girl 13 years old who had been losing sight for about two weeks and could count fingers only at fifteen feet. The retinal arteries were found extremely small, and the veins large with slight œdema about the disk. General examination elicited nothing of importance except that she was anæmic, had not menstruated, had had convulsions, and was neurotic. There were no stigmata of hysteria. Later a trace of albumin was found in the urine. Improvement began on the twelfth day but was not complete for a year. He attributes the convulsions, albuminuria, and retinal ischæmia to vasomotor disturbances in the various organs. Such cases, he remarks, which we call neuropathic, serve to take us back one link

farther along the chain of etiology toward the labyrinth of physiological chemistry for the ultimate explanation.

ALLING.

DOYNE (354, **Retinitis pigmentosa**) thinks that this disease has suffered more than others at the hands of those who "compile" treatises on the eye. He is particularly disappointed in finding nothing original in the work of Dr. W. T. Shoemaker on this disease. Doyne thinks there are several points which are fairly constant but have not been alluded to. Difficulty in diagnosis arises from its similarity to chorioiditis followed by retinal atrophy and pigmentation. Hemeralopia is generally the first symptom noticed by the patient. Torpor of the retina, or "slow night," may be found at an early stage. Images too are often slow to fade; there is prolonged persistence of the retinal image. In addition to "night blindness" there may be dazzling in bright light, a symptom usually associated with nyctalopia. Patients prefer good diffused daylight. But later they prefer a "*certain* artificial illumination." A very exact degree is chosen, neither too much nor too little. In one case bright moonlight was as useful as daylight. Another point noticed is a bright light which momentarily circles round the periphery of the field. Later in the disease ring scotomata are common. Central vision is preserved till late in the disease, but there are cases in which it fails early, sometimes producing nystagmus. Some cases do not advance beyond night blindness, the field remaining good. A lacelike deposit of pigment is found towards the periphery, but the extreme periphery escapes. Inside the pigmented zone may be seen distinctly, in many examples, another zone of grayish infiltration rather the color of powdered slate pencil, or perhaps paler. This can be seen in the retina and is especially associated with the vessels. The optic disk has a waxy appearance and the physiological cup is filled in. There is a peculiar opacity in the vitreous, an interlaced reticulum of fine opalescent threads just in front of the vitreous, like the gossamer seen on an autumn morning over a turnip field. This is very difficult to see. The same material may be present in "chunks" in the vitreous. Doyne alludes to an observation of Schobel in Norris and Oliver's *System* which illustrates a visible canal of Cloquet filled with

opalescent material. Schobel mentions that in three of five cases which he observed this canal was full of this very material which he has observed in the vitreous. The retinal vessels may become ensheathed in pigment, and from loss of retinal pigment the chorioidal vessels show up unduly. Doyne does not believe that the disease begins in the chorioid. There is a change in the capsule at the posterior pole of the lens which later develops into a posterior polar and ultimately a complete cataract. After extraction of the lens a recurrent film like a wrinkled capsule is apt to form. Heredity and consanguinity play an important rôle in the etiology of the disease.

Thickening of the retina is universal. Atrophy is not the main condition. There is an exudative process going on. Drusen bodies are not uncommon. Great improvement follows removal of the lens even when the cataract is not extensive. Retinal feeding is useful. T. HARRISON BUTLER.

BUTLER (355, **On the futility of the official tests for color blindness**) reports five recent cases in practice in which color blindness, congenital or acquired, could be overlooked if the Holmgren wool test alone be used, but could not escape detection by the use of the Edridge-Green lamp. He also lays stress on the desirability of frequent retesting of seamen and railway employes in order to find any acquired color blindness from such conditions as tobacco amblyopia or partial optic atrophy after retrobulbar neuritis.

A. H. PAYAN DAWNAY.

XXI.—OPTIC NERVE.

356. LANDOLT, MARC. **Varicosities of the papilla.** *Archives d'ophtalm.*, xxxi., p. 104.

357. GORDON, A. **Disease of the optic nerve as an early, or the earliest, symptom of multiple sclerosis.** *Archives of Internal Med.*, June, 1910.

358. FRIDENBERG, PERCY. **The nerve head in wood-alcohol amaurosis.** *Trans. Amer. Ophthalm. Soc.*, 1910.

359. REYE. **A contribution to tuberculosis of the optic nerve.** *Muenchener Medizin*, June, 1910, p. 1229.

360. DUBOIS. **Retrobulbar optic neuritis of nasal origin.** *Nederl. Tidschrift voor Geneeskunde*, 1911, i., No. 1.

361. SAENGER, ALFRED. **Choked disk in its relation to cerebral tumors and trephining.** *Jour. Amer. Med. Assoc.*, Sept. 24, 1910.

362. BARR, J. STODDART, and ROWAN, JOHN. **Optic neuritis and suppurative otitis.** *Brit. Med. Jour.*, 1910, i., p. 737.

LANDOLT (356, **Varicosities of the papilla**) observed, in the left eye of a soldier 63 years old, a very tortuous, threadlike anastomosis between a delicate infero-temporal artery and the larger supero-temporal branch, as well as a broad, tortuous connection, ending above in a diverticulum, between the two main veins, which were distended with blood. There was very probably an obstruction to the circulation in the course of the optic nerve, just behind the eyeball, between the papilla and the junction of the two principal branches of the central vein. Landolt has found no similar case described in literature. CAUSÉ.

GORDON (357, **Disease of the optic nerve an early symptom of multiple sclerosis**) has observed five cases in which the eye symptoms were very early manifestations of this disease. Out of fifty-six cases of multiple sclerosis he found eighteen, beside those mentioned, in which the optic nerve was affected either as a neuritis, retrobulbar neuritis, or optic atrophy. ALLING.

FRIDENBERG (358, **The nerve head in wood-alcohol amaurosis**) contends that a certain picture is characteristic of this condition—a deep excavation resembling a glaucomatous cup, a white atrophy, no evidence of connective tissue in the cup, but evidence of inflammation about the nerve in the form of perivascular striæ and pigment. He further suggests that the poison is not from alcohol *per se*, but from formalin or formic acid circulating in the blood as a result of incomplete combustion of methyl alcohol introduced into the system. ALLING.

REYE (359, **Tuberculosis of the optic nerve**) reports the case of a boy, 2 years old, who presented all the symptoms of a meningitis. He died on the second day, and the autopsy showed that the left optic nerve, the greater part of the chiasm, and the intracranial portion of the left optic nerve were transformed into a cheesy mass, surrounded by a layer of tuberculous granulation tissue of uneven thickness. Whether the focus originated metastatically, or the tuberculous meningitis had spread along the pia sheath of the vessels, was not determined.

Although the nasal condition was negative, DUBOIS (360, **Optic neuritis of nasal origin**) opened the sphenoid and pos-

terior ethmoid sinuses with good result, because the disease was constantly advancing and no other cause could be demonstrated. VISSER.

SAENGER (361, **Choked disk in its relation to cerebral tumors and trephining**), whose work along these lines is well known, believes that increased intracranial pressure is the cause of choked disk and that this opinion will be later universally accepted. The condition of the disk is an œdema, although there may be inflammation as a secondary manifestation. He draws conclusions from twenty-six cases of decompression. ALLING.

BARR and ROWAN (362, **Optic neuritis and suppurative otitis**) give the results of the examination of 160 cases of purulent middle-ear disease, and the authors draw the following conclusions:

1. Optic neuritis may occur in cases of purulent middle-ear disease without obvious signs of an intracranial complication.

2. Apart from optic neuritis, vascular changes of a lesser degree are frequent.

3. Cases of purulent middle-ear disease, in which optic neuritis or vascular engorgement of the fundus is present, are much less amenable to local treatment than those in which the fundus is normal.

4. As a general rule, an improvement in the eye condition is accompanied by improvement in the aural condition, while an increase in the intensity of the changes in the fundus or their persistence is associated with less amenability to treatment and greater gravity of the ear condition.

5. The most probable cause of vascular engorgement of the fundus or optic neuritis is serous meningitis (local or diffused).

6. Optic neuritis caused in this way is not usually followed by atrophy, and unless there are other symptoms demanding it, opening of the dura mater is unnecessary.

The authors consider that the cases in which these eye conditions persist should undergo the radical mastoid operation, and that those in which there is a tendency to subside may be subjected to more conservative treatment.

A. H. PAYAN DAWNAY.

XXII.—INJURIES, FOREIGN BODIES, PARASITES.

363. DUVAELE. **Asthenopia due to electric light.** *Soc. de médecine de Gand*, Jan., 1909.

364. KERRY, RICHARD. **Notes on some cases of trauma of the eye.** *Ophthalmology*, July, 1910.

365. WILDEY, A. G. **A case of optic atrophy following supraorbital injury.** *Lancet*, 1910, vol. ii., p. 1343.

366. JAMES, R. R. **Penetrating injury of the globe with panophthalmitis due to a gas-forming organism.** *Ophthalmic Review*, June, 1910.

367. BULL, C. S. **The post-operative history of eighteen cases of magnetic foreign bodies removed from the eye by the Haab or giant magnet.** *Trans. Amer. Ophth. Soc.*, 1910.

368. EVANS, J. JAMESON. **The estimation of the visual efficiency of injured workmen.** *Birmingham Medical Review*, Oct., 1910.

369. DE LIETO, VELLARA. **Contribution to the pathology of cysticercus in the vitreous, with particular regard to the plasma cells not hitherto described.** *Archivio d'ottalmologia*, 1909, Nos. 5 and 6.

370. DUPUY-DUTEMPS. **Subretinal cysticercus. Extraction.** *Annales d'oculistique*, 144, p. 305.

According to DUVAELE (363, **Asthenopia due to electric light**) six workmen, all blondes, were seized with so severe asthenopia after working a quarter of an hour in a room lighted by an arc light that they were obliged to stop work. Nothing was found at the examination to explain the asthenopia, and the only thing common to all the patients was the fact that they had been working in an atelier, lighted by an arc light. This light contained many ultraviolet rays. The use of yellow glasses prevented the trouble.

KERRY'S (364, **Notes on some cases of trauma of the eye**) cases are as follows: (1) A blood-staining of the cornea following a severe contusion. After fifteen months the cornea had cleared from the periphery and left only a faint stained area in the centre. (2) Hemorrhage probably between the posterior surface of the lens and vitreous, which was absorbed with perfect vision. (3) An eye inflamed and destroyed as the result of trauma and with subjective light sensations which stopped after enucleation. ALLING.

WILDEY (365, **A case of optic atrophy following supraorbital injury**) reports the case of a sailor who fell from a bicycle and sustained a wound over the right orbit with slight concussion. On recovering his senses he found that he was blind in the right eye; there was no proptosis or plus tension, and no change

in the fundus; sensation was unimpaired over the supraorbital area. Some perception of light had returned at the end of nine days; atrophic changes were observed a few days later in the right disk, and perception was absent from the nasal side of the fundus; the changes progressed until at the end of six weeks there was only perception of light in a small area in the lower and nasal portion of the 20° circle.

There were no obvious signs of fracture of the skull, but the optic atrophy was probably due to a fracture through the optic foramen. PAYAN DAWNAY.

JAMES (366, **Panophthalmitis due to a gas-forming organism**). A boy was shot in the eye with an air-gun. Next day there was early panophthalmitis and bubbles of gas were issuing from the wound. The eye was excised at once, and the boy made a rapid and complete recovery. Smear preparations showed large gram-positive bacilli, which proved to be anaërobic. A culture did not kill a guinea-pig but produced local necrosis of tissue. The micro-organism was *bacillus aërogenes capsulatus*. The feathered dart was found in the eye. T. HARRISON BUTLER.

BULL (367, **The post-operative history of eighteen cases of magnetic foreign bodies removed from the eye by the Haab or giant magnet**) records seventeen cases of successful extraction of foreign bodies from the vitreous or fundus. The extraction occurred through the original wound in seven cases, and through other channels in ten. Phthisis bulbi resulted in six and sympathetic ophthalmia in eight, enucleation being necessary in ten. The vision was permanently lost at the time of the accident in five. No permanently useful vision was gained in a single case, he having been particularly unfortunate in this respect. He believes it far wiser to make an incision in the sclera and remove the foreign body with the small magnet if possible. ALLING.

EVANS (368, **The estimation of the visual efficiency of injured workmen**) says that in the estimation of visual efficiency the following factors must be considered:

1. The visual acuity for distance and near.
2. The presence or absence of binocular vision.
3. The extent of the visual fields and the presence or absence of scotomata.

4. The condition of the ocular muscles—extrinsic and intrinsic.

5. Conditions of work.

6. The general—and especially the nervous—state of the patient.

As a rule, ***visual acuity*** is the most important factor in visual efficiency. Snellen's types should be used in all compensation cases. Illiterate types must be used when necessary. To circumvent malingerers, Evans has adopted the ingenious device of cutting up test types and pasting the letters on to a fresh card, arranging them at haphazard so that the malingerer cannot make up his mind to read down to a certain line. This can be done both with the ordinary types and with the trident E in different positions. The top E = $\frac{6}{60}$ is best omitted. Reversed types viewed in a mirror are also of great use. Jaeger's or Snellen's types are employed for near vision. Any great discrepancy between near and far vision should lead us to suspect malingering.

Industrial vision may be considered normal if vision is not less than $\frac{6}{12}$. It is generally held that if it falls below $\frac{6}{60}$ the individual cannot earn his living. Evans thinks this standard is too high.

Roughly speaking, he takes the following as a guide as to the vision required in different trades:

(*a*) For skilled workmen (engineers, fitters, mechanics, jewellers, machinists, etc.) and members of learned professions..... $\frac{6}{6}$ to $\frac{6}{24}$

(*b*) For unskilled workmen (laborers, miners, quarrymen, carters, etc.)..... $\frac{6}{12}$ to $\frac{6}{60}$

In looking through a list of a few hundred patients who have attended the Eye Hospital recently for various complaints which did not prevent them following their occupations, he found that the vision of those who were well able to follow skilled occupations is very often defective in one or both eyes. He mentions a few (errors of refraction corrected):

Blacksmith ..R.V. = $\frac{6}{12}$ L.V. = $\frac{6}{18}$ aphakia in left eye.

Nut and bolt maker ..R.V. = $\frac{6}{24}$ L.V. = $\frac{6}{24}$ glaucoma in each eye.

Engine-drivers	R.V. = $\frac{6}{6}$ L.V. = $\frac{6}{60}$; R.V. = $\frac{6}{6}$ L.V. = $\frac{6}{36}$.
Carpenter	R.V. = $\frac{6}{12}$ L.V. = $\frac{6}{12}$
Toolmaker	R.V. = $\frac{6}{24}$ L.V. = $\frac{6}{24}$
Tailors	R.V. = $\frac{6}{60}$ L.V. = $\frac{6}{12}$; R.V. = $\frac{6}{18}$ L.V. = $\frac{6}{18}$.
	R.V. = $\frac{6}{6}$ L.V. = $\frac{6}{60}$ L. squint.
Brass-finisher	R.V. = $\frac{6}{8}$ L.V. = $\frac{6}{36}$
Chandelier maker	R.V. = $\frac{6}{18}$ L.V. = $\frac{6}{12}$ nystagmus.
Compositors	R.V. = $\frac{6}{12}$ L.V. = $\frac{6}{12}$; R.V. = $\frac{6}{18}$ L.V. = $\frac{6}{18}$.
Bedstead maker	R.V. = $\frac{6}{60}$ L.V. = $\frac{6}{12}$.
Machinists	R.V. = $\frac{6}{18}$ L.V. = $\frac{6}{18}$; R.V. = $\frac{6}{24}$ L.V. = $\frac{6}{24}$.
Fitters	R.V. = $\frac{6}{30}$ (aphakia, etc.), L.V. = $\frac{6}{6}$; R.V. = $\frac{6}{6}$ L.V. = $\frac{6}{24}$ (cataract).
Electrician	R.V. = $\frac{6}{24}$ L.V. = $\frac{6}{18}$
Tram-driver	R.V. = $\frac{6}{18}$ L.V. = $\frac{6}{36}$ (conical cornea)
Needle-grinder	R.V. = $\frac{6}{12}$ L.V. = $\frac{6}{12}$
Cycle-polisher	R.V. = $\frac{6}{18}$ L.V. = $\frac{6}{18}$
Ironplate-worker	R.V. = $\frac{6}{18}$ L.V. = $\frac{6}{24}$.

Binocular Vision. There are few objective tests. Wetz's prism test is the best. This causes diplopia and the necessary movement to effect fusion can be detected. There are many well-known subjective tests. Haselberg's types are useful. Remy's diploscope and Harman's diaphragm test are useful. Roche's test is simple and demands no apparatus.

Pfalz's stereoscopometer may be employed if the surgeon wishes to have a special instrument for the purpose. But a one-eyed man can be an excellent mechanic; binocular vision is not necessary in most trades. It is bad policy for a man to give up his work because he has lost an eye.

Visual Fields. It is well before using the perimeter to make rough tests with the finger.

Slight diminution of the fields causes no reduction in the wage-earning capacity. Homonymus hemianopsia is more troublesome, and the reduction in wage-earning capacity may be estimated at as high a figure as 40%. But the nature of

the work must be considered. Central scotomata are serious, especially in railway work.

The paper considers muscular defects, aphakia, and the conditions of work. It is a very valuable contribution to ophthalmic literature and should be carefully studied.

T. HARRISON BUTLER.

XXIII.—OCULAR TROUBLES IN GENERAL DISEASE.

369. PARKER, W. R. **Report of two cases of chronic cyanosis with polycythæmia, including ophthalmoscopic examinations.** *Trans. Amer. Ophth. Soc.*, 1910.

370. COBBLEDICK, A. S. **Some ophthalmic conditions caused or influenced by diseases of the upper respiratory tract.** *Brit. Med. Jour.*, i., p. 1282.

371. PIKE, NORMAN H. **A case of cerebro-spinal rhinorrhœa with double optic atrophy.** *Ibid.*, i., p. 1104.

372. EDMUNDS, WALTER. **The treatment of Graves's disease with the milk of thyreoidless goats.** *Lancet*, 1910, i., p. 1135.

373. WHITE, W. HALE. **The treatment and prognosis of exophthalmic goitre.** *Lancet*, 1910, ii., p. 1599.

374. STURM, F. P. **A case of exophthalmic goitre cured by operation.** *Brit. Med. Jour.*, 1910, i., p. 1288.

375. WEBER, F. B. **Family amaurotic idiocy without characteristic ophthalmoscopic signs.** *Ophthalmoscope*, March 1, 1910.

376. TAYLOR, J. **Eye signs and symptoms associated with nervous diseases.** *Ophthalmoscope*, March, 1910.

377. BOYD, WILLIAM. **A case of tumor of the pituitary body.** *Lancet*, 1910, ii., p. 1129.

378. BRAMWELL, BYROM. **A case of intracranial tumor with alterations in the color fields.** *Lancet*, 1910, i., p. 631.

379. BARCK, C. **A case of brain abscess with rare ocular symptoms.** *Amer. Jour. of Ophthalmol.*, Nov., 1910.

380. WEISENBURG, T. H. **Exophthalmos in brain tumor.** *Jour. Amer. Med. Assoc.*, Dec. 3, 1910.

381. ZENTMAYER, WILLIAM. **A case of bitemporal hemianopsia with an unusual clinical picture.** *Annals of Ophthalmology*, Oct., 1910.

382. JAMES, R. R. **A case of hemianopsia.** *Ophthalmic Review*, Nov., 1910.

PARKER (369, **Report of two cases of chronic cyanosis with polycythæmia, including ophthalmoscopic examinations**) adds two cases to the list of this affection, which is characterized by chronic cyanosis, polycythæmia, and an enlarged spleen. The eye picture observed has already been described—engorged veins and some retina œdema. ALLING.

COBBLEDICK (370, **Some ophthalmic conditions caused or influenced by diseases of the upper respiratory tract**) draws attention to the possible relationship between diseases of the nose and mouth and corneal ulcer, dacryocystitis, and other septic conditions of the eye, and lays stress on the necessity of removal of such affections of the nose and mouth previous to operations on the eye. A. H. PAYAN DAWNAY.

PIKE (371, **A case of cerebro-spinal rhinorrhœa with double optic atrophy**) reports the following case. A patient at the age of 12 years had a severe illness with marked cerebral symptoms, lasting nearly twelve months, at the end of which she was quite blind. She continued to have attacks of headache and frequent epileptiform fits, the latter becoming less after eight years. When seen at 22 years of age she was found to have a double post-neuritic atrophy and to be without perception of light. She complained of a constant dripping of watery fluid from the right nostril, which had been going on for nine months, whilst the headaches had lessened and there had been only one fit. The fluid on analysis was found to contain globulin and a Fehling-reducing substance.

A. H. PAYAN DAWNAY.

EDMUNDS (372, **The treatment of Graves's disease with the milk of thyreoidless goats**) makes a further report concerning the cases under treatment, which were recorded in the *Lancet*, 1908, vol. i., and 1909, vol. i. Some cases show marked benefit from the treatment, the goitre being reduced, exophthalmos lessened, and the heart slowed. Three cases are also referred to under the care of Dr. Gilmour at the West Riding Asylum, two showing marked improvement in their mental symptoms, and the third a complete recovery with a diminished goitre and a pulse of 64.

PAYAN DAWNAY.

WHITE (373, **The treatment and prognosis of exophthalmic goitre**) has tried to follow up the history of all his cases of this condition in hospital and private practice during twenty years. As a result he finds that there is a natural tendency in many cases to recovery, even where treatment has not been persisted in; but judged from the standpoint of life assurance exophthalmic goitre patients are on the whole rather bad lives. As to the cause of death among the hospital patients,

gastro-enteritis was a frequent contributory, much less so among the private patients; in the two groups together several died with mental disease. With regard to treatment, none of this series of which the author writes was subjected to operation, the numerical results of which do not induce him to a very favorable view. Rest he considers the most important point in treatment, and next to it plenty of plain food; in some cases Moebius's antithyreoid serum has appeared to benefit, whilst in others no good has seemed to accrue; a like result occurred in the use of rodagen. PAYAN DAWNAY.

STURM (374, **A case of exophthalmic goitre cured by operation**) reports the following case. A woman, aged 32, for two years showed all the signs of Graves's disease: there was a large bilateral goitre, the neck measurement being twenty inches, and the pulse rate 120 to 140. All forms of medical treatment failed to afford relief, and surgery was resorted to. The right and larger lobe was removed under local anæsthesia, and recovery was uneventful; within a week the pulse fell to 80, and in a month the tremors ceased, and the exophthalmos lessened; seventeen months later all the symptoms of the disease had disappeared, and the patient was leading an ordinary busy life. He also refers to three other cases on which he had operated with good results.

A. H. PAYAN DAWNAY.

WEBER (375, **Family amaurotic idiocy without characteristic ophthalmoscopic signs**). The case was certainly one of family amaurotic idiocy possibly associated with internal hydrocephalus. The typical cherry-red spot was absent and there was no optic atrophy. The amaurosis was probably connected either with local changes in the retina or with alterations secondary to hydrocephalus. The amaurosis is probably due to diffuse changes such as have been demonstrated in the brains of these cases. The retinal lesions are not an essential part of the pathology.

T. HARRISON BUTLER.

TAYLOR (376, **Eye signs in nervous diseases**) groups the signs and symptoms of nervous diseases under four heads: pupillary; ocular movements; fields of vision; and fundus. Anisocoria as an isolated symptom has no significance. Reflex iridoplegia is the most important sign. The pupils are nearly

always unequal and the phenomenon may be unilateral. It is so frequent in tabes that those cases in which it does not occur are worthy of special study as regards etiology. It occurs in hemiplegia the result of endarteritis obliterans. The author has also seen it in severe muscular atrophy about the upper limbs. These cases reached a certain stage and then ceased to be progressive; they were probably due to a gummatous inflammation involving certain nerve roots. If the Argyll Robertson pupil be present and the case is not tabes there is always the probability that there is early general paralysis of the insane. It may be noted for years before serious symptoms supervene. The Argyll Robertson pupil invariably indicates infection with the *spirochæta pallida*, it is a parasyphilitic sign.

Diplopia is an early and common sign of disseminated sclerosis. Neuritis of the sixth nerve not uncommonly causes paralysis of the external rectus similar to Bell's palsy. Such a diplopia has no significance and rapidly clears up. Paresis of the sixth, however, necessitates a careful examination. It may be an early sign of tabes or of intracranial pressure. Paralysis of ocular muscles in alcoholic poisoning is of ominous import. Diplopia is a very common symptom in intracranial disease such as tumor.

The symptoms of *myasthenia gravis* should not be overlooked by ophthalmic surgeons. At first the only difficulty may be in keeping the eyes open; the ptosis is worse in the evening than in the morning. There is with the ptosis defective ocular movement in every direction. The muscles of the limbs have the characteristic myasthenic reaction to faradism. This disease can hardly be distinguished from chronic ophthalmoplegia due to slow degeneration of cells in the oculo-motor nuclei.

When nystagmus is present in insular sclerosis we can generally elicit a history of diplopia. Nystagmus or nystagmoid jerking is sometimes present in alcoholic peripheral neuritis.

A quadrantic defect in the visual field suggests a cortical lesion, especially should it be definitely quadrantic.

Not much importance can be attached to the homolaterality of tumor and most intense optic neuritis. Should optic neuritis be associated with sudden transient loss of vision, it

must be regarded as an ominous sign. Such patients suddenly die from respiratory failure. Tumor may be present without optic neuritis.

Primary optic atrophy is characteristic of tabes, and as a rule tabetic symptoms are absent. Even knee-jerks may be present for years. Optic atrophy may be an early sign of general paralysis.

The pale disks of disseminated sclerosis are often described as atrophic. Many such have perfect vision. Color sense is frequently impaired. The pallor is probably secondary to a retrobulbar neuritis. T. HARRISON BUTLER.

BOYD (377, **A case of tumor of the pituitary body**) describes the following case. The patient's illness started with neuralgic pains in the left temple, and then in the right; failure of sight began in the left eye eighteen months later, and in the right six months later still. The vision of the left was by this time hand movements and the field of vision in the right was hemianopic, the temporal half of the field being quite blind. In two years he started having epileptic fits without aura and sometimes passing into the status epilepticus. Next the left eye became totally blind, and during the following year the patient became ataxic and also had attacks of acute mania. Subsequently he became lethargic and lost the sight of the right eye; he was also getting very deaf. After eight years he was mentally dull, blind, partially deaf, not paralyzed in his limbs but with loss of sphincter control and altered reflexes; there was some weakness of the left superior and inferior recti and inferior oblique; ophthalmoscopically there were double optic atrophy and chorioiditis; there was a discharge of albuminous fluid from the left nostril. Remaining in this condition for a further four months he died suddenly. The post-mortem revealed a large tumor in the sella turcica which was extremely enlarged, its bony floor being destroyed and a communication with the left antrum of Highmore being established on the left side by which the cerebro-spinal fluid had escaped. Microscopically the tumor was a mixed-celled sarcoma. The cerebro-spinal fluid contained a large amount of albumin and showed excessive lymphocytosis; the lesion was shown to be non-syphilitic by its resistance to treatment and the subsequent microscopic examination. PAYAN DAWNAY.

BRAMWELL (378, **A case of intracranial tumor with alterations in color fields**) reports this case with notes of three others supporting the views of Bordley and Cushing (ARCHIVES OF OPHTHALM., 1909, p. 451) on the occurrence of alteration of color fields in this condition. The patient was a man, aged 56, who for two months had suffered from recurring painful cramps followed by painless clonus in the left calf, accompanied sometimes with loss of consciousness, but on one occasion when there was no such loss attended by a definite aura; finally there was complete loss of power in the left leg, and partial loss in the left arm. So far there was no eye affection. Three weeks later he had a febrile attack (100°.8 F.) with temporary headache and vomiting; the retinal veins were full, but there was no swelling of the disks. After another three weeks papillitis was present on both sides, more marked on the left; the fields showed constriction for white, also for yellow and blue, the field for red being less affected than for the two latter; the left yellow field was less than that of the right; there was also some interlacing of the fields for yellow, blue, and red. About a fortnight later the papillitis was much increased, the constriction of the fields was more marked, in some parts that of white being more reduced than that for the colors. The left arm was now completely paralyzed, the leg less so than two months previously; there was still absence of headache and vomiting. The patient was now operated on, a flap of bone being removed over the motor area; a hernia cerebri formed, and owing to the patient's behavior from mental derangement the wound became septic, and death ensued. At the post-mortem, beneath the hernia was found a large gliomatous tumor extending to the wall of the lateral ventricle and across the under surface of the corpus callosum slightly to the right side. The second case was that of a man, aged 41, with headache, vomiting, right hemiplegia, and aphasia; there was double optic neuritis and constriction of fields for white and colors but no interlacing. The third was a man, 22 years of age, suffering from headache, vomiting, and giddiness of one month's duration; there were also optic neuritis, constriction of fields but no interlacing, paralysis of the sixth nerve, and paresis of the seventh. The tumor was considered to be in the frontal lobe. The fourth was a woman aged 43; she had

fits, headache, and occasionally vomiting. Some of the fits were Jacksonian in character and affected the right arm. Optic neuritis developed on the left side; the fields showed right-sided hemianopsia with constriction for white and colors on the left side; there was no interlacing. The general condition of this patient improved under treatment with iodides, the disks and fields remaining as before.

PAYAN DAWNAY.

BARCK'S (379, **A case of brain abscess with rare ocular symptoms**) patient developed headache, vision greatly reduced, choked disks, and right homonymous hemianopsia with the dividing line passing through the fixation point. She had had two operations upon the mastoid. About nine months before, a decompression operation was performed but no abscess located although the headaches stopped and the choked disks were decidedly reduced. A month after this operation the patient died and an abscess was found in the posterior-superior portion of the temporal lobe. Twelve cases were found on record where abscesses of the temporal lobe produced hemianopsia. ALLING.

From the histories of seventy-five cases of brain tumor, WEISENBERG (380, **Exophthalmos in brain tumor**) relates eight in which exophthalmos developed. He draws the following conclusions: Exophthalmos accompanies brain tumors more frequently than is commonly supposed. It occurs only in those cases in which there is great intracranial pressure. It is likely to be produced when there is direct pressure on the cavernous sinus. When unilateral it would indicate the side of the lesion. ALLING.

ZENTMAYER (381, **A case of bitemporal hemianopsia with an unusual clinical picture**) reports the case of a patient who probably had a growth of the pituitary body of slow development. The blind part of the field regained vision after the lapse of many years. Only one case resembling it is on record. It is possible that the wearing away of the bone (sella turcica) relieved the pressure on the chiasm and allowed the nerve fibres to resume their function. ALLING.

JAMES (382, **A case of hemianopsia**). Hemianopsia occurring as an isolated complication of a head injury is rare. This subject has been much neglected in English and American

text-books and even treatises on neurology give only scanty references to the symptom. James gives a case of a woman who was thrown from an omnibus, falling on the right side of her head. Since the accident she could not see things on the right side of her head. The charts published show right homonymous hemianopsia, with slight general contraction.

T. HARRISON BUTLER.

OBITUARY

CARL HORSTMANN died on January 18, 1912, in his 65th year. He was First Assistant in the Berlin University Eye Clinic from 1874–1878, and since 1879 has been connected with the teaching staff in that University. For many years he had conducted the Report on Progress in the *Archiv für Augenheilkunde*, a very laborious task with difficulties usually not appreciated. This report appeared in translated form in these ARCHIVES. His contributions to ophthalmic literature have been on many clinical subjects. His genial personality endeared him both to his patients and to his colleagues.

BOOK REVIEWS

The price of COLLINS and MAYOU'S *Pathology and Bacteriology of the Eye*, published by Blakiston, is $4.00 and not $3.00 as stated in the book review of the last number.

The Pocket Atlas of the Fundus Oculi by JOHNSON and HEAD, which was favorably commented upon in the last number, is published by F. A. Hardy & Company, 10 South Wabash Avenue, Chicago, for America. Price $2.50.

X.—**The Prescribing of Spectacles.** By A. S. PERCIVAL. Second edition. Bristol: John Wright & Sons, Ltd., 1912. Price 5/6 net.

This excellent little manual now appears in a second edition. It has been revised, new paragraphs and two more tables added. The one table brings the values of various metre angles and the other the focal distances in inches and centimetres of different lenses. It is a very sensible and practical treatise.

A. K.

XI.—**Handbook of the Diseases of the Eye.** By SIR H. R. SWANZY and L. WERNER. Tenth edition, with illustrations. Pages 634. Philadelphia: P. Blakiston's Son & Company, 1912. Price, $4.00.

Swanzy's well-known text-book has now been brought thoroughly up-to-date. The text has been revised, many additions made, and the subject matter is thoroughly illustrated. The colored fundus drawings of Mr. Werner are excellent and are beautifully reproduced. The book in its present form takes its place amongst the best in the English language, and in its medium size is especially adapted for the use of medical students.

A. K.

FIG. 1. A. B., "DP," 26 years old, two years in Manhattan State Hospital, Wards Island. Left eye shows congestion and edema of entire disc, edges blurred and indistinct, filling in of physiological excavation. Veins dilated, dark and tortuous; relative size compared to arteries, $2\frac{1}{2}$ to 1. (Direct method of examination.)

FIG. 2. K. M., "DP," in Manhattan State Hospital for 13 months. Quite deteriorated. Right eye shows temporal pallor, with nasal congestion and edema, blurring of edges on the nasal side. Veins dark and tortuous, dilated; relative size of veins to arteries, 2 to 1. (Direct method of examination.)

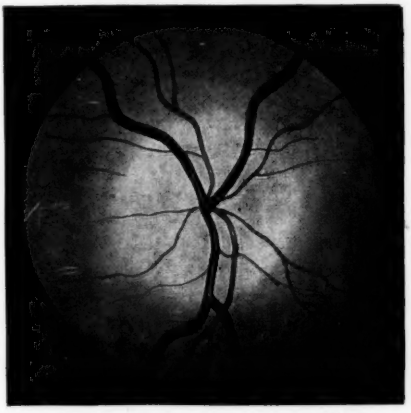

FIG. 3. M. E., "DP." Right eye. Shows pallor of entire disc. Edges blurred and indistinct. Veins dilated, dark and tortuous. Arteries slightly contracted. Relative size, compared to arteries, 2 to 1. (Direct method of examination.)

1.

2.